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FUNCTIONAL PULMONARY INCOMPETENCE, AND DILATATION AND ATHEROMA OF THE PULMONARY ARTERIES, AS COMPLICATIONS OF MITRAL STENOSIS.

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The occurrence of pulmonary regurgitation as a complication of mitral stenosis has interested me for some time, and I have in consequence carefully examined, from this point of view, all the cases of mitral stenosis I have seen. During the six years I was holding the post of Medical Registrar I had exceptional opportunities for carrying out these observations, having had access to all the cases of this nature admitted to the medical wards, and I take this opportunity of thanking my colleagues for allowing me to make use of the notes of them. I have collected sixteen cases bearing on this point, the largest collection I believe hitherto published. My interest in this condition was first aroused by Case 1. When she was admitted on the second occasion, in addition to a rumbling presystolic bruit at the point of the cardiac impulse there was "two inches to the inner side of the left nipple and traceable upwards into the third left space,

a whistling early diastolie bruit." Three explanations were suggested to explain the occurrenee of this bruit—

(1) That it was due to the mitral stenosis.

(2) That it was due to aortic regurgitation.

(3) That it was due to pulmonary regurgitation.

I must eonfess that I first of all favoured one of the first two views. Dr. Goodhart subsequently saw the ease, confirmed the presenee, charaeter, and position of the bruit, and definitely stated that in his opinion the murmur was due to pulmonary regurgitation, the result of a dilated pulmonary artery. This opinion was verified by the post-mortem examination whieh I performed on Mareh 14th, 1893. The pulmonary artery was deeidedly thickened and dilated, it measured 101·6 millimetres in circumference, the average eircumferenee of the pulmonary artery in females being about 88 millimetres.

Adams recorded two eases of mitral stenosis in which dilatation of the pulmonary artery was found. In the first case it is thus described: "The pulmonary artery was unusually dilated." In the seeond ease: "The pulmonary trunk was dilated nearly to twiee its natural size." These are two of the earliest cases I have been able to find recorded.

Stokes deseribes a case of dilatation of all the cavities of the heart and of the pulmonary artery and the aorta, but makes no mention of pulmonary regurgitation from dilatation of the pulmonary artery, as a eomplication of mitral stenosis.

To Graham Steele belongs the chief credit for drawing attention to this form of pulmonary incompetence. In his work entitled, "The Physieal Signs of Cardiac Disease," when writing of the regurgitant murmur resulting from dilatation of the aorta, he says, "I am inclined to believe that a murmur of similar mechanism occurs on the right side of the heart when there is much obstruction to the pulmonary eirculation with a dilated pulmonary artery." The same physieian in 1889, in an article entitled, "The Murmur of High Pressure in the Pulmonary Artery," writes, "I wish to plead for the admission among the auscultatory signs of disease of a murmur due to pulmonary regurgitation, such regurgitation oeeurring independently of disease or deformity of the valves, and

as the result of long continued excess of blood-pressure in the pulmonary artery." He points out that in cases of mitral stenosis an early, soft blowing, diastolic bruit following or immediately running off from an accentuated pulmonary second sound, may be heard at the sternal ends of the third and fourth intercostal spaces. If the second sound happens to be reduplicated the murmur usually follows the latter sound. This bruit is not usually persistent at first and shows great variableness in its intensity, but the accentuation of the second sound is always present.

Barr, when writing of the bruit associated with mitral stenosis, says, " The early diastolic murmur may be purely mitral in origin, but I am convinced that it is often confounded with a short, soft blowing, diastolic murmur, which not infrequently occurs in this disease at the moment of closure of the pulmonary valve, and which arises from slight regurgitation into the right ventricle owing to the high pulmonic tension." And later, writing of the pulmonic second sound he says, " In a certain number of cases there is a short, soft, diastolic murmur (to which I have before alluded) in the pulmonic area, due to slight regurgitation through the pulmonic orifice. This is especially apt to happen when the right ventricle is dilated and does not completely empty itself during systole, the high tension in the pulmonic system drives a certain amount of blood back through its closing valves into the unemptied ventricle. Some think this impossible, as the valve is usually found competent at the autopsy, but there is a remarkable difference between the pressure of one or two inches of water and the pulmonic tension in a case of mitral stenosis."

Sansom says, the early diastolic murmur associated with mitral stenosis when there is no other sign except the murmur, is indicative of aortic regurgitation, and he criticises Graham Steele's explanation that they are due to regurgitation into the right ventricle in consequence of the extreme tension within the pulmonary artery caused by the mitral obstruction. He quotes Percy Kidd, who objects to this view of Graham Steele's on the grounds that if the murmur were really due to this cause there would be some evidence of leakage or valvular defect found not infrequently at the necropsies made on cases of mitral stenosis. Sansom thus

writes, "I am not aware of a single case that has demonstrated either structural defect or passive dilatation permitting regurgitation through the valves of the pulmonary artery, and I, therefore, regard the theory as unproven. It is also in my opinion improbable, seeing that such diastolic murmur is, so far my experience goes, unknown in other conditions—very grave mitral regurgitation for instance—when the pulmonary artery is subject to severe internal pressure. Moreover, if it were true, I think we ought to be able to trace in dilatation of the pulmonary artery the replacement of the loud pulmonic sound heard in cases of mitral stenosis and mitral regurgitation by a diastolic murmur, or the affixing of such a murmur to the exaggerated normal sound. I have never met with, nor have I heard of any such experience." He considers the diastolic murmur to be due to either the mitral lesion itself or to aortic regurgitation. I do not consider that these arguments are conclusive, for the pulmonary artery is a highly elastic structure, and it is quite reasonable to suppose that as soon as the patient dies and the increased pulmonary tension is relaxed, the dilated pulmonary artery, if moderately dilated, contracts down to its normal size. One of the chief characteristics of the bruit is its variability, and this may be explained by a disappearance of the dilatation under favourable conditions. Case 16 illustrates this point, for the bruit was heard on March 17th and 20th, but not on the 23rd or subsequent dates. There is, however, conclusive evidence to show that in some cases of mitral stenosis there is appreciable dilatation of the pulmonary artery.

Duckworth has reported a case of tricuspid and mitral stenosis in which symptoms of pulmonary incompetence were present. The patient was a married woman, æt. 23. A diastolic murmur was heard in the pulmonary area but subsequently disappeared. At the post-mortem examination the pulmonary valves were not markedly altered. The mitral valve was much stenosed and button-hole in shape. There was decided dilatation of the pulmonary artery. "The pulmonary arterial reflux was probably explicable by the dilated state of the vessel and the disappearance of the murmur was coincident with increasing low blood-pressure, as the vital powers failed towards the last."

I have divided my cases into two groups, the first nine in which post-mortem examinations were made, the last seven in which the diagnosis of the condition rested on the clinical evidence alone.

The post-mortem evidence of dilatation of the pulmonary artery, then, is as follows :—

Case 1. The pulmonary artery appeared to be dilated, and it felt much thicker than normal, almost as thick as the aorta (Drs. Goodhart and Shaw confirmed this). The pulmonary orifice measured 91·5 millimetres, and 20 millimetres above the orifice the circumference of the vessel was 101·6 millimetres. In addition to this I noted that the pulmonary valves appeared to be very thin and loose, and each corpus arantii was considerably thickened. The free edges of these valves were slightly curled towards the ventricle. There were no recent vegetations, and the valves were thin and loose. It was evident that these valves had not been involved by any previous attack of endocarditis. The measurements showed considerable dilatation, not only of the pulmonary artery but of the orifice itself.

Case 2. The pulmonary artery appeared to be much larger than normal, for it seemed almost double the size of the aorta. The pulmonary orifice measured 8 centimetres and 12 millimetres above the orifice the circumference of the lumen was 10·16 centimetres. Although the measurements do not point to much increase in size, the appearance of the vessel at the time of the post-mortem certainly indicated a considerable amount of dilatation, and allowing for a certain amount of contraction after death, when the tension had relaxed, it would seem that during life there must have been considerable dilatation.

Case 3. The pulmonary artery was thickened and dilated; it was almost as thick as the aorta; 2·5 centimetres above the pulmonary valves it measured 8·25 centimetres in circumference when laid open.

Case 4. In this case there was undoubted post-mortem evidence of dilatation of the pulmonary artery. Dr. Perry described the condition as follows : "On the right side the pulmonary valves

measured 11·4 centimetres in circumference, and, therefore, there was doubtless pulmonary regurgitation."

Case 5. The pulmonary orifice measured 7 centimetres, just above the orifice the artery measured 8·8 centimetres, and had the appearance of being considerably dilated and was much larger than the aorta. In this case again the actual measurements did not show much dilatation, but the appearance of the pulmonary artery and its size when compared to the aorta certainly pointed to an abnormal distension of that vessel.

Case 6. The pulmonary orifice measured 7·6 centimetres. The pulmonary artery was dilated, and was much larger than the aorta.

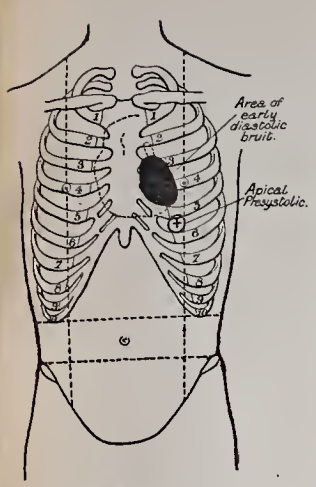
Case 7. The pulmonary orifice measured 7·6 centimetres. The pulmonary artery was dilated and was much larger than the aorta.

In cases 6 and 7, therefore, although the measurements did not show actual dilatation, a comparison with the aorta showed that the pulmonary artery in each case was undoubtedly abnormally large.

Case 8. The pulmonary artery was much larger than the aorta, was much thicker, and had atheromatous patches in it. It measured 7·6 centimetres across, against 5·7 centimetres of the aorta. In this case also, although the measurement was not great, there was undoubted dilatation of the pulmonary artery.

Case 9. The pulmonary artery looked very large, the valves measured 8·9 centimetres, and were normal in appearance.

In cases 10, 11, 12, 13, 14, 15 and 16 the evidence of pulmonary incompetence from dilatation of the pulmonary artery rested on clinical evidence alone. Very strong evidence can be adduced of increased tension in the pulmonary arteries in cases of mitral stenosis by post-mortem examination. I have never performed or seen performed a post-mortem examination on a case of advanced mitral stenosis without finding thickening, dilatation and atheroma of the branches of the pulmonary arteries in the lungs. Very little attention, if any, is drawn to this change in the branches of the pulmonary arteries in the descriptions given in the various text-books, of the backward pressure effects of chronic



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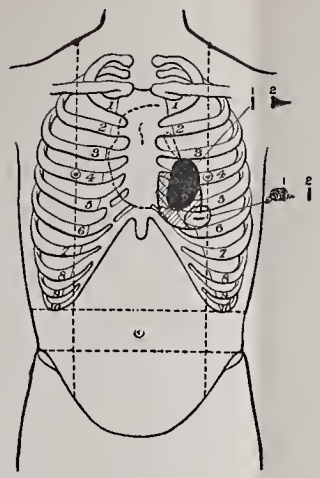

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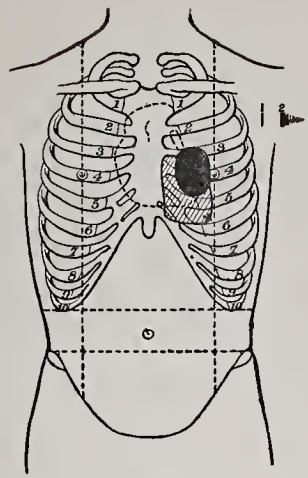

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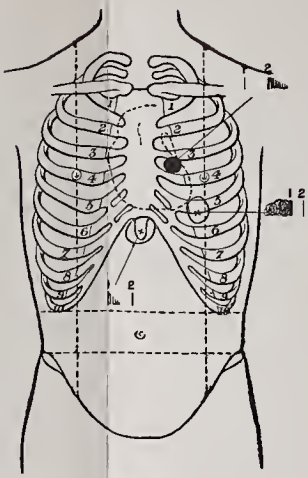

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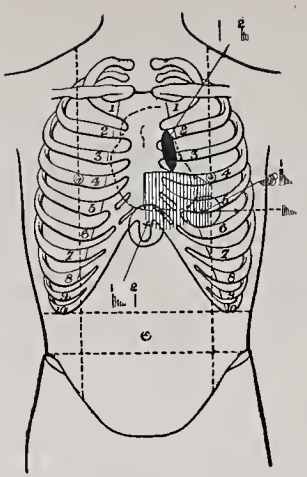

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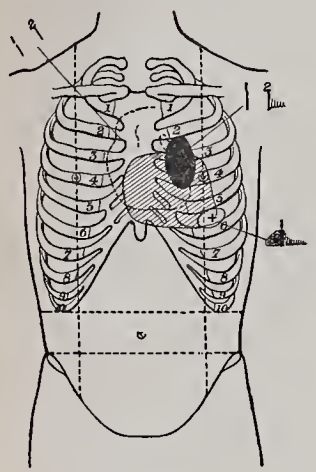

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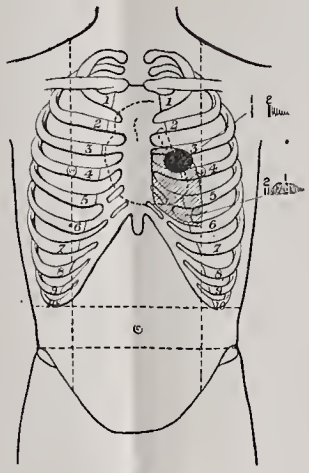

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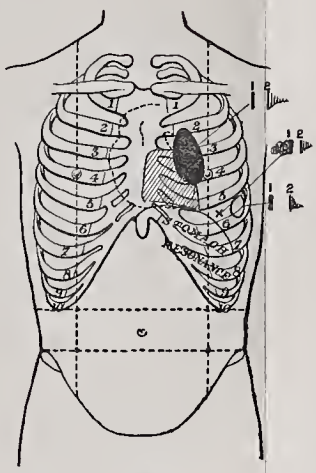

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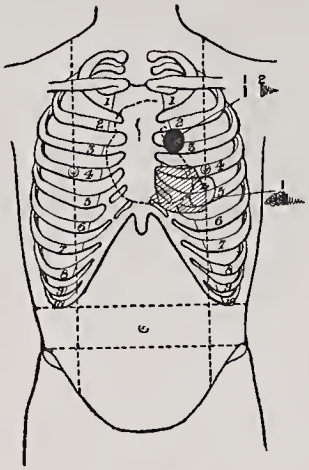

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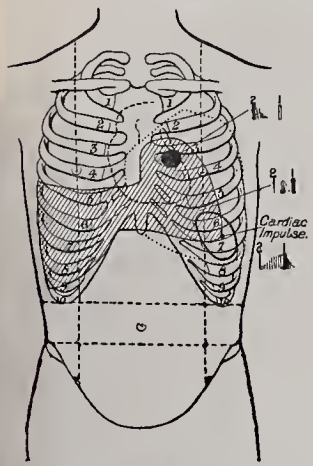

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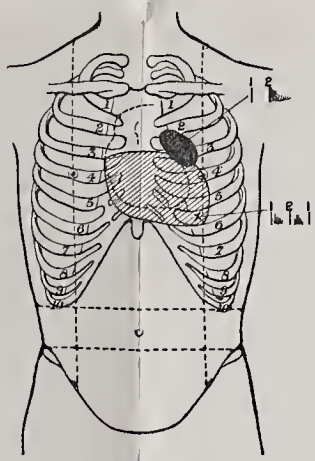

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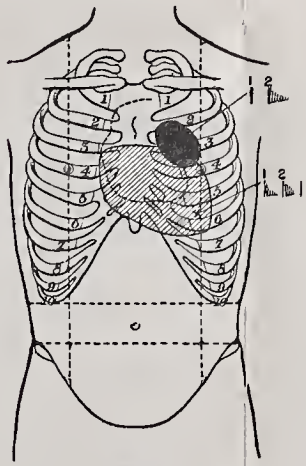

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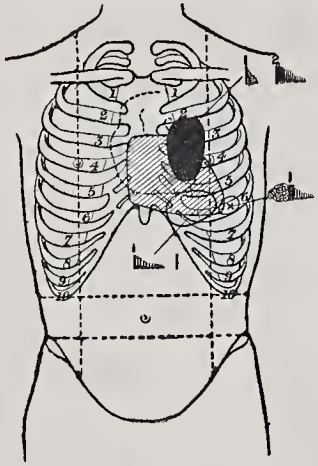

Fig. 13.

valvular disease of the left side of the heart. I claim that these changes in the pulmonary arteries should find an important place in all descriptions of the effects of backward pressure on the other organs of the body from chronic valvular disease of the heart.

Hilton Fagge drew attention to this point. He wrote: "The increase of tension in the pulmonary vessels soon leads to changes in their walls, which become thickened and hypertrophied. In the main trunks of the pulmonary artery this is particularly noticeable. The records of post-mortem examinations at Guy's Hospital contain notes by Dr. Moxon of the case of a boy, aged ten years, in whom the coats of the pulmonary artery were nearly twice as thick as those of the aorta at its thickest part, and less striking examples of the same are very commonly met with. The artery also becomes greatly dilated." Fagge also states that another important result of this increased tension is an atheromatous change in the branches of this vessel, and he quotes a striking example of this condition, recorded by Dr. Conway Evans.

It appears that the first observer to call attention to these changes in the pulmonary arteries was Dittrich, who described it as occurring particularly in the smaller branches, and looked upon this change as being the actual cause of the pulmonary apoplexies which are so frequently met with under such conditions.

Yeo has published a case of atheroma and dilatation of the pulmonary arteries secondary to mitral stenosis and aortic disease. "The pulmonary artery from the semilunar valves down to the very first subdivision is irregularly dilated and inelastic, the inner coat throughout being studded with hard prominent yellow patches, some of which are rough on the surface and look like ordinary atheromatous ulcers." The mitral valve was much stenosed. He considered the change to be due to the increased tension and consequent mechanical irritation.

Pepper also reports a case of sclerosis and atheroma of the pulmonary arteries secondary to mitral stenosis.

Whittaker writes: "Sometimes the valves are perfectly sound, but the orifice is dilated, so that the condition is that of relative insufficiency. The cause in these cases is usually an arterio-

sclerosis which produces dilatation of the pulmonary artery and the cause of the arterio-sclerosis is for the most part syphilis."

I quite agree that the valves may be perfectly sound and that the orifice may be dilated and so produce a relative insufficiency. I also agree with the fact that arterio-sclerosis is usually associated with this condition, but I do not consider there is sufficient evidence to show that syphilis has any direct connection with it. There is, however, conclusive evidence that increased tension is the mort important causative factor, for I maintain that it is more frequently observed as a complication of mitral stenosis than of any other condition, but it is also associated with other chronic valvular lesions of the left side of the heart and with pulmonary emphysema. I have only seen one case in which no obvious cause was found which could have given rise to increased pulmonary tension.

Mott, when writing on the important influence of internal strain as a factor in the production of arterio-sclerosis says: "The relative infrequency of affection of the pulmonary artery which occurs, indeed, only under such conditions as involve increased tension of its walls, as, for example, prolonged mitral stenosis, indicates the importance of internal strain as a factor in the degenerative process.'

Osler writes: "Sclerosis of the pulmonary artery is met with in all conditions which for a long time increase the tension in the lesser circulation, particularly in mitral valve disease and emphysema.

Sometimes the sclerosis reaches a high grade and is accompanied with aneurysmal dilatation of the primary and secondary branches, more rarely with insufficiency of the pulmonary valve."

The description of the condition of the pulmonary arteries in the cases in which post-mortem examinations were made is as follows:—

Case 1. The branches of the pulmonary arteries in the lungs were atheromatous, more especially on the right side and in the right lower lobe.

Case 2. The pulmonary arteries were thickened, dilated and atheromatous, and stood out prominently from the cut surface of the lungs, with open lumina.

Case 3. The two main branches of the pulmonary artery were thickened and atheromatous, and the small branches in the lungs were thickened, atheromatous, and dilated, and stood out prominently from the cut surface of the lung, with open lumina.

Case 4. In the pulmonary artery were atheromatous patches, and in the left branch of it was a small piece of ante-mortem clot.

Case 5. The branches of the pulmonary arteries in the lungs were thickened and atheromatous, standing out prominently, with open lumina from the cut surface of the lung.

Case 6. On section (of the lungs) the pulmonary arteries stood out very prominently from the cut surface; they were dilated, very much thicker than normal, showing marked atheromatous changes, and appearing in fact almost as thick as similar sized bronchial tubes.

Case 7. The intima of the pulmonary artery was thickened and the larger branches showed marked atheromatous changes.

Case 8. The pulmonary artery had atheromatous patches in it.

Case 9. The pulmonary branches were greatly dilated and the coat showed irregular cracks in the intima, which were blood-stained. These appeared to be due to over distension; there was no atheroma.

In all the cases, therefore, in which a post-mortem examination was made there was very strong and conclusive evidence of strain and increased tension in the pulmonary arteries. Dilatation, thickening and atheromatous patches were the changes most frequently noticed. In none of the cases was there any evidence of syphilis.

Hunter was the first to point out that the pulmonary sigmoid valves do not "do their duty" so well as the aortic valves. He proves this by experimentally injecting the arteries towards the ventricle. Gibson has made some interesting experiments on the pulmonary valves of the dead heart with a column of fluid consisting of a solution of sodium chloride of specific gravity 1050 at 98·5 F., and to quote his words the following is the

average result of his series of experiments: "From the semilunar valves of the pulmonary artery of the sheep a strong jet escaped until the column of fluid measured fourteen and a half inches from which height it trickled until the valves became competent with a column of nine inches. In the ox, a strong jet was emitted down to twelve inches and dropping of the fluid reduced the superincumbent column to the height of six inches, when competence was established. In the healthy human heart a jet escaped down to thirteen inches, and the valves were competent with eight inches of fluid resting on them. Now, in each case of the pulmonary valves with a column of fluid six feet in height perfect competence was obtained in a very simple manner by constricting the pulmonary artery. A cord tied round the artery exactly at the attachment of the valves gave the means of perfect control over the escape, so that by varying the amount of tightening, the jet was converted into a drop falling quickly or slowly and this in turn was totally stopped. The whole diminution of circumference amounted to a few lines. This shows clearly that the escape is caused by distension of the elastic artery and relative incompetence of the valves." It was found that the aortic valves treated in a similar manner allowed of no escape, and the experiments showed that the pulmonary orifice is not closed so perfectly as the aortic, and point to the fact that any abnormal pressure in the pulmonary artery may give rise to reflux of blood into the right ventricle.

The marked structural changes found in the pulmonary arteries may therefore be looked upon as the result of the increased tension produced in the pulmonary circulation by the obstruction to the flow of blood through the mitral orifice. Mitral stenosis produces hypertrophy and dilatation of the left auricle, and hypertrophy and dilatation of the right ventricle. It would be difficult to imagine, therefore, that the pulmonary artery, which is situated between these two hypertrophied and dilated structures, would escape being damaged. That it does not escape is shown by the changes reported in the accounts of the post-mortem examinations in the first nine cases. It is a well known fact that when there is dilatation of the first part of the aorta, a reflux of blood occurs

into the left ventricle, and this may happen without there being any pathological change in the valves themselves. It has been shown by experiment that the pulmonary orifice is less resistant than the aorta, so that it is not difficult to imagine that the pulmonary artery and its orifice may become dilated as a result of the enormously increased tension produced by mitral stenosis, and may give rise to a functional incompetence without any actual disease of the valves.

There is also post-mortem evidence of this dilatation and incompetence, and I refer in particular to cases 1 and 4. I again repeat that the absence of post-mortem dilatation is not a conclusive proof that there was no dilatation during life. The elasticity of the artery, as soon as the increased tension was relaxed, unless the vessel happened to be enormously and permanently stretched, would assert itself, and the vessel would reassume its normal size.

Newton Pitt states that pulmonary incompetence is the rarest of all the valvular lesions of the heart, seventeen cases only were noticed during a period of twenty-three years in 11,000 necropsies. He gives a list of causes based on an analysis of 115 cases, and mentions that of this number twelve were due to dilated pulmonary artery.

In nineteen out of twenty-one cases of dilated pulmonary artery mitral stenosis was the cause. Pitt definitely concludes that there is both clinical and pathological evidence that the pulmonary arteries may become dilated in advanced mitral stenosis. He also states that in nearly all the cases post-mortem evidence of any structural change at the orifice will be absent.

The Condition of the Mitral Valve, Aortic Valves, Right Ventricle and Tricuspid Valves.

In all the cases in which post-mortem examinations were made the mitral valve was much stenosed and changes were also noted in the left auricle and right ventricle.

Case 1. The mitral orifice was very small, only just admitting the tip of the little finger; it was funnel shaped. The valve was much thickened and it measured 23·8 millimetres. The chordæ

tendiniæ were shortened and thickened. The aortic valves were healthy. The left auricle was hypertrophied. The right ventricle was hypertrophied and its wall measured 13 millimetres in thickness. The tricuspid valves were healthy.

Case 2. The heart weighed 397 grammes. The mitral valve was very thickened and calcareous, and the orifice would only just admit the tip of an index finger, it measured 3·81 centimetres in circumference; it was button-hole in shape. The chordæ tendiniæ were thickened and shortened. The aortic valves were normal. The left auricle was hypertrophied and dilated, and there was an ante-mortem thrombus in the appendix of the left auricle. The right ventricle and auricle were dilated, and the wall of the right ventricle was hypertrophied. The tricuspid orifice measured 10·16 centimetres.

Case 3. The heart weighed 350 grammes. The mitral valve was thickened and calcareous, and its circumference measured 3·17 centimetres. There were a few recent vegetations on the auricular edge of this valve. The chordæ tendiniæ were short, thickened, and adherent to each other. The aortic valves were healthy. The left auricle was hypertrophied and dilated. The right ventricle was hypertrophied, its wall measuring 9·5 millimetres in thickness. The right auricle was a little dilated. The tricuspid orifice measured 10·4 centimetres.

Case 4. The heart weighed 482 grammes. The mitral orifice was much stenosed and would just admit the index finger. The aortic valves were normal. The aortic orifice measured 6·35 centimetres. The tricuspid orifice measured 15 centimetres. There were ante-mortem thrombi in the appendices of both auricles.

Case 5. The mitral valve was thickened and calcareous, its orifice was much narrowed and measured 5 centimetres in circumference. The chordæ tendiniæ were thickened and shortened. The aortic valves were thickened and adherent but appeared to be competent. The left auricle was hypertrophied and dilated. The right ventricle measured 11·45 centimetres across at the base, and its wall was hypertrophied. There was ante-mortem thrombosis of the appendix of the left auricle.

Case 6. The heart weighed 510 grammes. The mitral valve was hard and thickened, and the mitral orifice would only just admit the tip of the little finger; it measured 37 millimetres in circumference. The chordæ tendiniæ were shortened and thickened. The aortic valves were healthy. The left auricle was hypertrophied. The right ventricle was much hypertrophied, and measured two centimetres in thickness. The right auricle was hypertrophied and dilated.

Case 7. The heart weighed 510 grammes. The mitral orifice was much narrowed and thickened and was button-hole in shape. The orifice would only just admit the tip of the little finger. The chordæ tendinæ were shortened and thickened. The aortic orifice measured 5·6 centimetres; the valves were certainly thickened but were quite competent. The left auricle was considerably dilated and hypertrophied, and the endocardium was much thickened. The right ventricle and auricle were hypertrophied and dilated. There was ante-mortem thrombosis of the appendix of the right auricle. The tricuspid valve was shortened and thickened and its circumference was 12·7 centimetres.

Case 8. The heart weighed 397 grammes. The mitral valve was converted into a calcareous ring, just admitting the end of the index finger; there were a few vegetations on the edge. The edges of the aortic valve were thickened and rolled. It seemed as if there must have been some regurgitation, but no murmur was ever heard, and the left ventricle was not much enlarged. The right ventricle and auricle were dilated and hypertrophied. The tricuspid valve had thickened edges and allowed regurgitation.

Case 9. The mitral valve was stenosed; It measured 7·6 centimetres. The aorta was dilated, and the valves were thickened and doubtfully competent. The right ventricle was much hypertrophied. The tricuspid valve was thickened, and it measured 14·3 millimetres.

An analysis of the above shows that the weight of the heart was noted in six of the cases, the lowest weight recorded was 350 grammes and the highest 510 grammes, so that considerable enlargement was common.

The mitral valve was small in all the cases; the smallest measurement was 23·8 millimetres and the greatest 76 millimetres. It was thickened in seven of the cases and calcareous in four, so that it was evident that the changes were of old standing.

In five of the cases the aortic valves were described as being healthy or normal. In two, they were described as thickened but competent, and in two as thickened and doubtfully competent, but in both of these last mentioned cases there was no clinical evidence of either aortic or pulmonary incompetence.

In four of the cases the left auricle was described as being hypertrophied and dilated and in two as being hypertrophied. In eight, the right ventricle was mentioned as being hypertrophied, and in case 6, its wall measured 20 millimetres in thickness. In three, the tricuspid valves were thickened; the smallest measurement of the tricuspid orifice was 10·16 centimetres and the largest 15 centimetres. In two of the cases antemortem thrombi were found in the appendix of the left auricle; in one in the appendix of each auricle and in one in the appendix of the right auricle. The above changes all point to the enormous backward pressure which existed as a result of the marked stenosis of the mitral orifice in each case.

An analysis of all the post-mortem examinations on cases of advanced mitral stenosis would show almost identical changes, and one of the most extraordinary features of this disease is the comparative rareness of evidence of dilatation of the main trunk and orifice of the pulmonary artery.

The explanation, I repeat, must be found in the elastic recoil of the vessel when the tension is relieved by death or cardiac failure.

It must be evident that as mitral stenosis leads to hypertrophy of the right ventricle, the right ventricle must have much more work to do. If the right ventricle, then, has more work to do, and does it, there must be a corresponding increase in the tension of the pulmonary artery and its branches. That this is so, is shown by the changes I have already described in the branches of this vessel in the lungs. The effect appears at first to be produced on

the smaller branches, but later on, when the tension gets beyond a certain point, the main trunk and orifice yield to the excessive tension and pressure, and so the vessel becomes dilated and reflux of blood occurs into the right ventricle without there being any actual lesion of the pulmonary valves.

The Physical Signs and Symptoms of Functional Pulmonary Incompetence.

The most important physical sign of this condition is the presence of an early diastolic murmur. The position and character of this murmur will be best manifested by analysing the sixteen cases on which this paper is based, and by investigating the statements made by other observers.

Case 1 :—

(*a*). A whistling, early diastolic murmur was heard two inches to the inner side of the left nipple, and it was traceable upwards into the third left space. The bruit became fainter as it was traced upwards.

(*b*). An evenly sustained, diastolic, humming bruit of some harshness in quality, and commencing directly after the second sound and continuing nearly, if not quite, through the pause. The bruit occupies chiefly the right side area, and it is much louder immediately below the pulmonary valves (Dr. Goodhart) (*vide* fig. 1).

Case 2 :—

(*a*). A well marked blowing, early diastolic murmur was heard in the third and fourth left spaces, midway between the left border of the sternum and the left nipple line (*vide* fig. 2.) In the aortic area the second sound was loud and clear in character.

(*b*). In the pulmonary area, about three quarters of an inch outside the left border of the sternum, and over the third and fourth intercostal spaces, a distinct, soft, early blowing diastolic murmur following a rather loud second sound was heard (*vide* fig. 3). In the aortic area the second sound was sharp, and ringing in character.

Case 3 :—

(*a*). A diastolic bruit was heard in the fourth left space, with reduplication of the second sound in the third left space.

(*b*). A faint, short diastolic bruit was heard in the third left space, coming immediately after a very accentuated second sound (*vide* fig. 4).

(*c*). In the second and third left intercostal spaces, just outside the left border of the sternum, an early diastolic murmur was heard (*vide* diagram 5).

Cases 4, 5, 6, 8 and 9. There was no clinical evidence of pulmonary incompetence.

Case 7. The second sound was very much accentuated in the second left space, where also a systolic and a faint diastolic murmur could be heard.

Case 10. In the second and third left intercostal spaces, midway between the left border of the sternum and the left nipple line a soft blowing, early diastolic murmur was heard (*vide* fig. 6). The aortic second sound was sharp, clear and accentuated.

Case 11. A faint, soft, blowing early diastolic murmur could be heard in the third left space, about two centimetres from the left border of the sternum, and 1·5 centimetres internal to the left nipple line (*vide* fig. 7). The aortic second sound was clear and distinct, and there was no visible pulsation of the carotid arteries.

Case 12. In the second and third left intercostal spaces midway between the left border of the sternum and the left nipple line, a soft early diastolic murmur was heard (*vide* fig. 8). No bruits were heard in the aortic area.

Case 13. A loud and accentuated second sound, followed by a soft, blowing, early diastolic murmur was heard in the second left space, just outside the left border of the sternum. The aortic sound was sharp and clear and no bruit could be heard accompanying or following it (*vide* fig. 9).

Case 14. In the third left space near the sternum the first sound was reduplicated, the second sound was loud and was followed by a faint, blowing, diastolic murmur (*vide* fig. 10).

Case 15:—

(*a*). A faint, soft, diastolic murmur was heard in the second left space, and could be traced downwards and outwards for about three centimetres (*vide* fig. 11).

(*b*). At the junction of the second left costal cartilage, and traceable downwards and outwards half-way towards the left nipple, a short, soft, diastolic murmur was heard (*vide* fig. 12).

Case 16. A diastolic murmur was heard in the second and third left intercostal spaces, just outside the left border of the sternum, and inside the left nipple line (*vide* fig. 13).

Pitt states that the most important signs of pulmonary incompetence are: Pulsation in the second and third left spaces close to the sternum, a condition which is partly the result of dilatation of the infundibulum and pulmonary artery, and a diastolic murmur audible on the left side of the sternum.

Balfour states that "In all cases of pulmonary diastolic murmur hitherto recorded there has always been, I believe, a loud systolic murmur preceding it." This is not my experience with the pulmonary diastolic murmur produced by functional incompetence of the valves resulting from dilatation of the pulmonary artery and orifice. Systolic basal bruits were only heard in two of the sixteen cases. It might be argued that if the pulmonary artery is dilated the condition necessary for the production of a fluid vein would be present and there should be a systolic bruit. But the probable reason of the absence of such a systolic murmur is that the pulmonary orifice is also dilated as well as the artery, so that the conditions for a fluid vein do not actually exist.

Sansom describes the bruit of pulmonary regurgitation as best heard over the second left intercostal space, and says it may be audible down the sternum to the apex of the left ventricle.

Broadbent describes the murmur of pulmonary regurgitation as being best heard in the third left intercostal space and that it may be conducted downwards.

Gibson describes the bruit of pulmonary incompetence as follows: "The murmur may be diffused over a wide area, but it appears invariably to have its maximum intensity in the second

left space. It is propagated towards the apex of the heart and its line of conduction is chiefly to the left of the sternum. The character of the murmur varies considerably. It may be soft and blowing or harsh and rasping." He records three cases of functional pulmonary incompetence.

In the first case (M.G., æt. 18), a short, sharp, high pitched, soft, diastolic bruit was heard immediately following the second part of the reduplicated second sound. It was heard over a small triangular area, two and a quarter inches by two inches along the lower border of the sternum from the lower border of the third costal cartilage to the upper border of the fifth costal cartilage. The murmur was quite different in character to the murmur of mitral stenosis, and there were no signs of aortic disease, so that it was looked upon as being a murmur indicating regurgitation from the pulmonary artery into the right ventricle "due to the increased pressure and consequent dilatation of the orifice, with relative and transient incompetence of the cusps."

In the second case (N. W., æt. 16) there was a diastolic murmur, soft in character, heard best in the third left intercostal space; the point of the maximum intensity was one and a half inches from the mid sternal line. The murmur followed a very distinct second sound. It could only be traced a short distance in each direction. There was no capillary pulsation; the pulse was not splashing in character. At the post-mortem examination the mitral valve was much stenosed. The pulmonary orifice was much larger than the aortic. The right ventricle was much enlarged. The aortic valves were competent.

In the third case (A. L., æt. 19), the first sound in the pulmonary area was pure, the second sound was much accentuated and was followed immediately by a short, soft, rather high-pitched diastolic murmur, quite different in character to the diastolic bruit audible in the mitral area. The point of the maximum intensity of the bruit was at the sternal end of the third left intercostal space, it could be traced a little upwards, but no bruit was heard in the carotids. In this case there was no evidence of aortic regurgitation.

Barie classifies the causes of pulmonary incompetence thus :—

A. True pulmonary incompetence due to actual disease of the valves.

B. Functional or relative incompetence, in which its valves are not diseased, the incompetence being purely functional and due to dilatation of the pulmonary artery, this latter form being much rarer than the former.

Barie mentions that Pawruski and Gouget have recorded cases of functional incompetence of the pulmonary artery in some cases of mitral stenosis. He thus writes when describing the physical signs of this condition : " Le signe capital est fourni par l'auscultation elle décèle la présence d'un souffle diastolique, le long du bord gauche du sternum dans le deuxième espace intercostal gauche.

Ce bruit " semblable de tous points a celui qu'on recontre dans les cas ordinaires d'insuffisance aortique." (Stokes). " Se propage dans la direction de l'artère pulmonaire c'est a dire le long du sternum jusqua vers le 4° espace intercostal. On la vu se propager encore jusqua vers la base de l'appendice xiphöide, à la façon de certaines insuffisances aortique ; dans un autre cas on le retrouvait même dans la rêgion interscapulaire."

Bramwell describes pulmonary incompetence as being attended by a diastolic murmur, heard in the pulmonary area best, traceable downwards and to the right, and often heard at the lower end of the sternum.

Gerhardt states that the diastolic bruit of pulmonary incompetence is appreciably augmented during expiration on account of the increased intra-ventricular tension.

From the analysis of the eleven cases in which a murmur was audible, it will be seen that the most characteristic physical sign of the condition, is, an early diastolic murmur immediately following the second sound. The second sound may be accentuated or reduplicated. The bruit is usually soft and blowing in character, it is often faint, it may be short, evenly sustained, or may continue nearly through the diastolic pause. It may be humming or whistling in character (*vide* case 1).

A glance at the diagrams will give the best idea of the distribution of the bruit, and one fact in particular becomes evident, viz., that the bruit is generally best heard not close to the left border of the sternum, as is usually stated, but midway between the left nipple line and the left border of the sternum. This is of great importance, and is probably one of the reasons why this complication of mitral stenosis is not more frequently recognised, as this particular region is not examined so systematically as it might be. If no bruit is heard in the second, third or fourth left intercostal spaces close to the sternum, the region just outside this area, which is midway between the left border of the sternum and the left nipple line, is frequently not examined at all, and so the bruit may be overlooked.

The distribution of the bruit was as follows: In the second and third left intercostal spaces five times, third and fourth left intercostal spaces three times, third left intercostal space three times, second left intercostal space once, and second, third and fourth intercostal spaces once. If the position of the bruit in each individual intercostal space is analysed the result is, third left intercostal space twelve times (out of thirteen), second space seven times, fourth space four times. From this it will be seen that the bruit is most frequently heard in the third left space, midway between the left sternal line and the left nipple line.

The pulmonary second sound was noted as being loud or accentuated in eight of the cases, and in two of the cases the second sound in the pulmonary area was reduplicated. The aortic second sound was described as being loud, clear or normal in six of the cases; in one it was faint.

The cardiac dulness was increased in thirteen of the cases, and this can be best seen by looking at the diagrams. The increase of dulness in most of the cases was upwards and to the right, indicating hypertrophy of the right ventricle.

The position of the cardiac impulse was dependent on the amount of hypertrophy and dilatation of the right ventricle. The character of the impulse did not present any peculiarity which would help in making a diagnosis of pulmonary incompetence.

Visible pulsation in the pulmonary area was only noted twice (cases 1 and 2). A diastolic thrill was felt in the third left intercostal space once (case 1). A bulging of the chest in the precordial area was noted in four of the cases.

The pulse did not present any peculiar diagnostic character. The rate varied from 74 to 136, the average being 107. It was described as compressible 9 times, regular 6, irregular 4, full 3, soft 2, dicrotic 2, weak 2, and intermittent 2. It was never described as being splashing or collapsing in character in any of these cases. Marked pulsation of the carotid and other arteries was absent.

Albuminuria was noted in	8	of the cases.
Râles were heard at the bases in ...	8	"
Cyanosis was noted in	7	"
Œdema of the Legs was noted in ...	6	"
Enlargement of the Liver was noted in	5	"
Epigastric Pulsation was noted in ...	4	"
Pleuritic Effusion was noted in ...	4	"
Ascites was noted in	3	"
A troublesome cough was noted in ...	2	"
Hæmoptysis was noted in	2	"
Hæmaturia was noted in	1	"

From the above it will be seen that there are no symptoms or signs which are characteristic of functional pulmonary incompetence, with the exception of the early diastolic bruit which is audible most frequently in the third left intercostal space, and can be best heard at a point midway between the left border of the sternum and the left nipple line.

Diagnosis.

A diagnosis of functional pulmonary incompetence can only be made after a most careful physical examination. There are no symptoms which are peculiarly diagnostic of this condition. Aortic regurgitation is the most likely lesion to be confused with it, and I shall therefore compare and contrast the symptoms of these two conditions.

A patient suffering from aortic regurgitation is usually pale, whereas a patient suffering from functional pulmonary incompetence is cyanosed. In eight out of eleven cases cyanosis, especially of the lips, was noticed.

The pulse.—In aortic regurgitation the pulse is usually splashing or collapsing in character. In pulmonary incompetence the pulse exhibits no special peculiarity, in fact, it is most likely to be characteristic of advanced mitral stenosis, as pulmonary incompetence does not usually occur unless the stenosis of the mitral valve is extreme and the condition is of long standing.

In aortic disease a characteristic feature is the marked visible pulsation of the carotid, temporal, brachial and other superficial arteries. No such marked or excessive pulsation is noticeable in pulmonary incompetence. In aortic regurgitation capillary pulsation can usually be demonstrated, but it is not present in cases of pulmonary regurgitation.

The position of the cardiac impulse does not help to distinguish these conditions. I have constantly noticed that when mitral stenosis and aortic regurgitation are associated together, the left ventricle does not hypertrophy to such an extent as it does when aortic regurgitation is the only lesion present, so that both in cases of aortic regurgitation and mitral stenosis, and of pulmonary incompetence and mitral stenosis, the position of the cardiac impulse will depend chiefly on the degree of hypertrophy of the right ventricle. If there is any difference it is slight, the impulse perhaps being a little lower and further to the left in the case of long continued aortic regurgitation and mitral stenosis on account of the slightly larger size of the left ventricle.

Palpation may reveal in the case of functional pulmonary incompetence a pulsation in the second and third left intercostal spaces, a condition which would not be likely to be associated with aortic regurgitation. Aortic regurgitation might be due to dilatation of the first part of the aorta, but in this case there would be pulsation in the second and third right intercostal spaces close to the right border of the sternum. It would, however, be most unusual to find aneurysm of the first part of the aorta associated with mitral

stenosis. A thrill is not common with regurgitation from either orifice.

The cardiac dulness in cases of pulmonary incompetence, secondary to mitral stenosis, is usually increased upwards and to the right, indicating considerable hypertrophy of the right ventricle, the increase of the dulness in these directions is much more likely to be associated with pulmonary than with aortic incompetence.

The fact of a diastolic bruit being heard on the left side of the sternum is put forward by some observers as an important indication of pulmonary rather than of aortic incompetence. The text-books are very misleading on this point, for the early diastolic bruit indicative of aortic regurgitation is almost invariably described as being best heard in the third right intercostal space close to the sternum. I admit that this is so in a small proportion of the cases of aortic regurgitation, but I maintain that in nearly 90 per cent. of the cases the bruit is best heard in the third *left* intercostal space close to the left border of the sternum, and my experience is that the diastolic bruit of aortic regurgitation is not only best heard, but is first heard, in the third left space close to the left border of the sternum, and immediately beneath the lower border of the third left costal cartilage.

In pulmonary regurgitation an early diastolic bruit is heard best in the third left space, not close to the left border of the sternum, but midway between the left border of the sternum and the left nipple line. In all cases of advanced mitral stenosis this area should be most carefully auscultated for the early diastolic bruit of functional pulmonary incompetence.

The bruit of functional pulmonary incompetence is usually softer and less distinct than that of aortic regurgitation.

The aortic second sound in cases of pulmonary incompetence may be heard to be clear and distinct, whereas the pulmonary second sound is followed or replaced by the bruit.

In aortic regurgitation the bruit may be propagated into the carotid arteries, but in pulmonary incompetence no bruit is audible over these vessels.

Gerhardt has pointed out that the murmur of pulmonary incompetence is increased in intensity during expiration. This test was unfortunately not examined for in the cases I have reported.

Another observation which I cannot confirm or refute, is, that in pulmonary regurgitation an interrupted vesicular murmur may be heard during inspiration at the angle of the right scapula.

Broadbent writes: "Before venturing on a diagnosis of pulmonary regurgitation, it must be ascertained not only that the pulmonic sound is impaired, but also that the carotid throb and collapsing pulse are absent, and that the aortic second sound is unimpaired. No special train of symptoms can be attributed to pulmonary regurgitation."

Functional pulmonary incompetence must also be distinguished from pulmonary incompetence due to actual lesions of the pulmonary valves. Pulmonary incompetence from all causes is the rarest of all the valvular lesions of the heart. The pulmonary valves are hardly ever, if ever, affected by rheumatic endocarditis, so that, if, in a case of advanced mitral stenosis there is an early diastolic murmur indicative of pulmonary incompetence, it may be looked upon as being due to functional incompetence of the valves from dilatation of the pulmonary artery and its orifice. If, however, there is pyrexia and other physical signs and symptoms pointing to infective endocarditis, the probability will be that there are vegetations on the pulmonary sigmoid valves, rendering them incompetent. In connection with infective endocarditis affecting the pulmonary valves, I have observed, that in three out of the five cases which I have seen, there was undoubted evidence of gonorrhœa.

Some physicians are of opinion that this early diastolic bruit which is occasionally audible in the second, third and fourth left intercostal spaces is due to the mitral stenosis itself. I consider this interpretation is most unlikely and unsatisfactory, for I have never heard the presystolic or mid-diastolic bruits produced by mitral stenosis in these spaces and I fail to see why mitral stenosis should be considered capable of producing an early diastolic murmur in this position. Mitral stenosis without doubt is very occasionally indicated by an early diastolic murmur at the

point of the cardiac impulse, and between the impulse and the left border of the sternum, but there does not seem any justification for interpreting the early diastolic murmur which is audible most frequently in the third left space midway between the left border of the sternum and left nipple-line as being the direct result of the mitral lesion. That this murmur is the result of dilatation of the pulmonary artery and functional incompetence is a much more satisfactory explanation, and it is borne out by the very definite morbid changes which are found in the pulmonary artery and its branches in necropsies on cases of advanced mitral stenosis.

Conclusions.

1. That as a result of extreme mitral stenosis there is enormously increased tension produced in the pulmonary arteries.

2. That as a result of this increased pulmonary tension, the smaller branches of the pulmonary arteries in the lungs become dilated, hypertrophied and atheromatous.

3. That as a further result of this increased tension in the pulmonary artery, the main artery may become dilated and so may its orifice also.

4. That as a result of this dilatation of the pulmonary artery and its orifice, a reflux of blood may occur into the right ventricle through functional incompetence of the valves.

5. That the chief clinical indication of this functional pulmonary incompetence, is, the occurrence of an early diastolic murmur on the left side of the upper part of the chest, and, that the point where the bruit is best and most frequently heard, is, in the third left intercostal space midway between the left border of the sternum and the left nipple line.

6. That although there may be this clinical evidence of pulmonary incompetence, the post-mortem examination may fail to show any incompetence of the valves or dilatation of the pulmonary artery.

7. That the fact of there being no actual post-mortem evidence of regurgitation in some of the cases is no proof that the incompetence did not exist during life.

8. That the explanation of the last condition is as follows:—That the elasticity of the pulmonary artery has not been permanently injured by the increased pulmonary tension, and that as soon as the tension is relieved the vessel contracts to its normal size.

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LIST OF CASES.

Case 1.—C. S., æt. 36, music mistress, was admitted February 25th, 1891, under the care of Dr. Taylor (clerk, A. Leigh Allworth) for weakness and cough. There was a history of typhoid fever at sixteen years, and scarlet fever at twenty-one years. Since the typhoid fever she had always been short of breath, and in December, 1890, she had been compelled to give up her work.

Condition on admission, 25th February, 1891.—The cardiac impulse was in the fifth space, three-quarters of an inch internal to the nipple; the first sound was accentuated both at the apex and base, and the second sound reduplicated. There was a faint murmur with the first sound at the apex. Fine râles were heard on both sides of the chest, but the resonance was good. On March 2nd, 1891, the impulse was felt in the fourth and fifth spaces, and extended a little beyond the nipple-line. The cardiac dulness was not increased. A presystolic bruit was heard all along the fifth space, from about one and a half inches external to the nipple-line to the epigastric angle; it ended in a loud abrupt first sound. The second sound was accentuated in the second left space, and was also well heard at the apex, where it had a peculiar, short clicking character, rather difficult to describe. The first sound in the fourth space was short and sharp. The diagnosis was mitral stenosis, and she left the hospital on March 16th, 1891, much relieved. (P.S. 116, 92. Clerk, L. Slater.)

She was readmitted under Dr. Pye-Smith on the 27th April, 1892. The cardiac dulness was limited above by the upper border of the fourth rib, and externally by the nipple-line. Internally it reached nearly to the mid-line of the sternum. The cardiac impulse was in the fourth and fifth spaces, and was felt as far out as the nipple-line. There was a very long, loud, rasping, presystolic bruit, audible best in the fourth space, three quarters of an inch internal to the nipple and there was also a thrill. Some râles were heard at the bases of the lungs. The urine contained a small quantity of albumen. *Mitral stenosis* was again diagnosed. A day or two before she went out *Dr. Pye-Smith heard a faint diastolic murmur just to the left of the sternum below the third space.* The heart-sounds were otherwise the same, and the thrill was still present. Presystolic pulsation in the vessels (? veins) of the neck was also noted. She left the hospital on May 23rd, 1892. (G. 122, 1893, clerks, H. Hewetson, L. A. Parry, A. K. Matthews.)

She was readmitted under Dr. Goodhart on December 6th, 1892. The cardiac impulse was felt in the same situation as in April, and there was a diastolic thrill. Over the point of impulse a rumbling presystolic bruit was heard, running up to the first sound. *Two inches to the inner side of the left nipple, and traceable upwards into the third left space, a whistling early diastolic bruit was heard.* This bruit became fainter as it was traced upwards. There were troublesome fits of coughing, with occasional slight hæmoptysis, and moist râles were heard at the bases of the lungs. The urine contained albumen.

On December 9th, the following note was made in the report by Dr. Goodhart:—"She is rather blue about the lips and ears, suggesting mitral

stenosis. There is on palpation a grating first sound at the apex, followed by almost a click for the first sound, and then a faint diastolic thrill is perceptible; but as one passes up to the base, there is a much more pronounced systolic impulse over the third interspace to the left of the sternum, and after it a well-marked diastolic thrill. On auscultation at the normal impulse, there is characteristic presystolic bruit, short, sharp and grating, terminating in the first sound, the faint thrill being represented by a distant diastolic But on passing upwards to the base, one comes finally upon a second loud, evenly-sustained diastolic humming bruit, of some harshness in quality, and commencing directly after the second sound, and continuing nearly, if not quite, through the pause. On carefully mapping out the area of the bruit, although it is not quite confined to it, yet it occupies chiefly the right side area, and it is much louder immediately below the pulmonary valves."

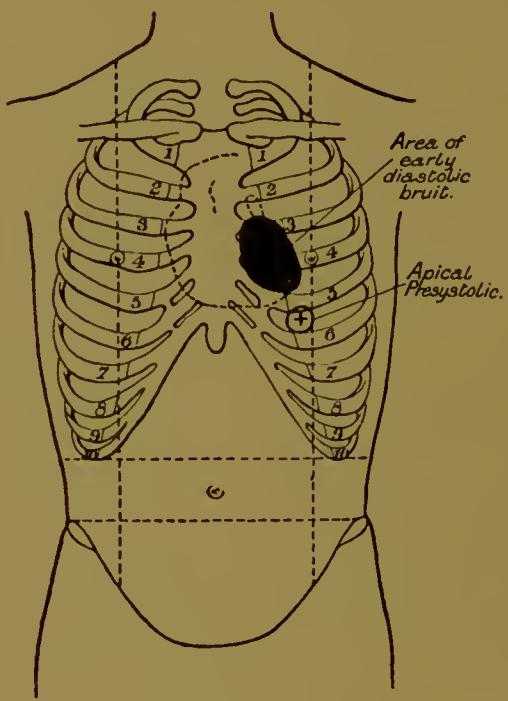

Fig. 1.

A few days later, after ascertaining the observations made at previous admissions, Dr. Goodhart made another note that the previous observations seemed to point to the physical signs then present being produced by a dilated pulmonary artery.

Signs of pleurisy were found soon afterwards on the left side, and on January 23rd 426 cubic centimetres of clear serous fluid were drawn off from the left side of the chest. The fluid was *acid*, specific gravity 1011, and contained twenty-four parts per 1000 of albumen.

The patient improved considerably after this, and left the hospital on February 23rd, 1893, the bruits remaining the same.

She was readmitted on March 8th, 1893, for bronchitis and vomiting.

Condition on admission.—Pulse 90, respiration 30, temperature 99°, a diastolic thrill was felt over the cardiac impulse in the fifth space, and also over the pulmonary area. On auscultation at the apex, there was a loud bruit, commencing almost at the beginning of diastole, and running quite up to the first sound. A very loud and high-pitched diastolic murmur was heard in the pulmonary area. Râles and rhonchi were heard on both sides of the chest. There was no œdema of the face or feet. The urine was loaded with urates, specific gravity 1030, and contained five parts per 1000 of albumen. She died suddenly on March 13th, 1893.

Post-mortem, 102, 1893, by J. H. Bryant.—The body was wasted, and the face had a haggard expression. The brain was not examined. The thyroid gland was healthy, but there was some extravasation of blood around the lower part of the neck at the level of the lower border of the thyroid. The pleura over the lower lobe of the left lung was thickened, opaque, and greyish-white in colour. 340 cubic centimetres of fluid were found in the right pleural cavity, and 280 cubic centimetres in the left. Both lungs were red, tough, and firm from chronic congestion (red induration). The lower lobe of the left lung was compressed, of a dull, slate-grey colour, and readily sank in water. *The branches of the pulmonary arteries in the lungs were atheromatous, more especially on the right side and in the right lower lobe.* The larynx, trachea, bronchi, and bronchial glands were healthy. The pericardial sac contained about 30 cubic centimetres of serous fluid. The heart was not weighed (it being left with the lungs to form a museum specimen). The right ventricle was empty, and there was a little post-mortem clot in the right auricle. The right side of the heart was enlarged, and the wall of the right ventricle much thickened, measuring 13 millimetres. The muscle was of good colour, and the tricuspid valves were healthy. *The pulmonary valves appeared to be very thin and loose, and each corpus arantii was considerably thickened. The free edges of these valves were slightly curled towards the ventricle.* The walls of the left auricle were a little hypertrophied, and the endocardium was thickened. The mitral orifice was very small—only just admitting the tip of the little finger. The mitral valve was funnel shaped, the opening being at the apex. The valve was much thickened, the chordæ tendinæ were shortened and thickened, and some of the musculi papillares were directly continuous with the edge. It measured 23·8 millimetres. The aortic valves were healthy. On opening the pericardial sac, *the pulmonary artery appeared to be dilated, and it felt much thicker than normal—almost as thick as the aorta (Drs. Goodhart and Shaw confirmed this). The pulmonary orifice measured 91·5 millimetres, and 20 millimetres above the orifice; the circumference of the vessel was 101·6 millimetres.* On tracing up the branches in the lungs, a number of patches of atheroma were seen, especially on the right side and in the vessels of the middle and lower lobes. There was some atheroma of the thoracic and abdominal aorta. The liver weighed 994 grammes, and was soft; it showed no sign of backward pressure. The spleen was small and hard, it weighed 76·2 grammes. The kidneys weighed 203 grammes. The capsules stripped off easily, but the surfaces were scarred and irregular. The uterus was large, and the cervical canal large and patent. There was a cyst the size of a pigeon's egg in the left ovary. The right ovary was bound down by old adhesions to the broad ligament. The remaining viscera were healthy.

Case 2.—F. N., æt. 40, under the care of Dr. Taylor (clerk, L. C. Martin) was admitted on December 27th, 1897, for dropsy and dyspnœa. Her father and mother were alive and healthy. One sister died of dropsy. When fifteen years of age she had an attack of rheumatic fever. She did not remember any other serious illness, but she stated that she had attacks of palpitation when ten years old. In September, 1896, she first noticed swelling of her thighs, followed by swelling of her legs and ankles. Rest in bed relieved her, and she remained fairly well until June, 1897, when she was admitted into Mary ward under Dr. Pye-Smith on June 30th.

Condition, July, 1897 (P. S. 223).—*Circulatory system:* pulse 88, regular, soft and compressible. The cardiac impulse was visible in the fifth left space just internal to the nipple-line; it was diffuse in character and could be seen two inches internal to the nipple-line. No thrill was felt. Dulness, *vide* diagram. At the impulse a short rumbling presystolic murmur running up to a loud first sound, which was followed by a faint systolic murmur, was heard. *In the third and fourth left spaces (vide diagram) a well-marked*

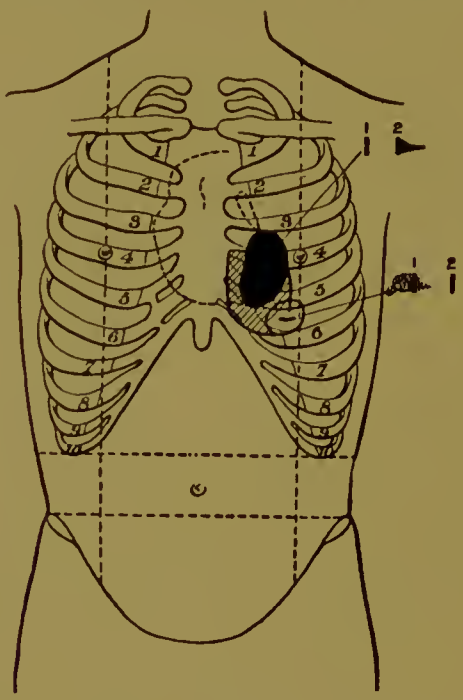

Fig. 2.

blowing early diastolic murmur was heard. In the aortic area the second sound was loud and clear in character. There was no visible pulsation of the carotids or other arteries.

Condition on admission, December, 1898.—Temperature 98°, pulse 110, respiration 30. Anæmic. She was suffering great pain and was unable to pass her urine. Her legs were very swollen and œdematous. *Circulatory system.*—Pulse 110, regular, soft and compressible. The cardiac impulse was diffuse in character and best visible in the fifth and fourth spaces just internal to the left nipple line. Pulsation was also visible in the third left space above and

in the second left space close to the sternum. A retracting impulse in the epigastrium synchronous with the carotid beat was also noted. The above mentioned pulsations were confirmed by palpation. No thrill was felt. The cardiac dulness was limited above by the lower border of the third rib, internally by the left border of the sternum, below by the fifth rib, and externally by a line drawn from the left nipple to the impulse. At the apex a loud systolic murmur was heard, traceable outwards into the left axilla, and nearer the sternum a rumbling diastolic murmur running close to the first sound but not quite up to it. *In the pulmonary area about three-quarters of an inch outside the left border of the sternum and over the third and fourth spaces* (vide diagram) *a distinct, soft, early blowing diastolic murmur,*

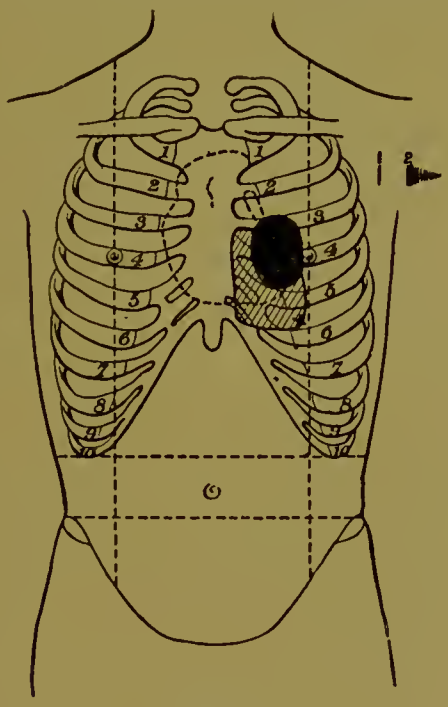

Fig. 3.

following a rather loud second sound was heard. In the aortic area the second sound was sharp and ringing in character. The abdomen was distended with fluid. Râles were heard at both bases behind. The urine contained a considerable amount of albumen. December 29th, paracentesis abdominis to ten litres was performed. January 1st, she was very dyspnœic and cyanosed. Heart sounds as described above. On January 5th, about 8.30 p.m., she died suddenly.

The *post-mortem* examination by J. H. Bryant, was made sixteen hours after death. There were old pleural adhesions over the posterior surfaces of both lungs, especially over the right, where they were firm, thick, and fibrous. Both lungs were œdematous and in a condition of red induration. Recent infarcts were found in both lungs. *The pulmonary arteries were thickened, dilated and atheromatous, and stood out prominently from the cut surface of the lungs, with open lumina.* Ante-mortem thrombi were found in several

of these vessels. The pericardium was healthy. The heart weighed 397 grammes. There was a thin layer of fat over the right ventricle. The coronary arteries were thickened and atheromatous. The right ventricle was full of post-mortem clot. A large ante-mortem thrombus was found in the appendix of the right auricle. The right ventricle and auricle were dilated, and the wall of the right ventricle was hypertrophied. The tricuspid orifice measured 10·16 centimetres. The valve was not thickened. *The pulmonary artery appeared to be much larger than normal, for it seemed almost double the size of the aorta. The pulmonary orifice measured 8 centimetres, and 12 millimetres above the orifice the circumference of the lumen was 10·16 centimetres.* The pulmonary valves were not thickened, and appeared normal. A large ante-mortem thrombus was also found in the appendix of the left auricle. The left ventricle and auricle were hypertrophied and dilated. The endocardium of the left auricle was much thickened. The mitral valve was very thickened and calcareous and the orifice would only just admit the tip of an index finger; it measured 3 81 centimetres in circumference; it was buttonhole in shape. The chordæ tendiniæ were thickened and shortened; some calcareous process from the valve extended into the muscle of the left ventricle. *The aortic valves were normal.* The stomach, œsophagus, and intestines were congested. 1847 cubic centimetres of clear yellow serous fluid was found in the peritoneal cavity. The *liver* weighed 1474 grammes. The surface was slightly irregular and showed numerous patches of perihepatitis, and on section it presented an advanced nutmeg condition. *Pancreas* very hard. *Spleen*, 226 grammes, very hard. *Kidneys*, 340 grammes, scarred and showing recent yellow infarcts.

Case 3.—E. S., æt. 41 years, sack sewer, was admitted 15th May, 1895, under the care of Dr. Goodhart (clerk, R. B. Stamford), for shortness of breath and swelling of legs. She was a widow, and had never had any children, but had had one miscarriage. She had always worked very hard. She had been in the hospital seven times before, chiefly for cardiac trouble. Her first admission was under Dr. Pavy, in 1883, for intestinal obstruction, and while under treatment she developed her first attack of rheumatism.

She was readmitted under Dr. Goodhart, February 8th, 1888, and remained in the hospital until March 20th, 1888. She then had a presystolic bruit and thrill at the apex, and she had rheumatic pains while in the hospital. The diagnosis made was mitral stenosis and bronchitis. (G. 117, 1888. Clerk, J. V. Blackford.)

She was readmitted January 11th, 1889, under Dr. Taylor, and remained in until January 30th, 1889. There was the same presystolic bruit and thrill, and in addition, she had a reduplicated second sound in the pulmonary area. Mitral stenosis and pleurisy were diagnosed. (C. 171, 1889. Clinical, J. M. Gill.)

Her next admission was May 30th, 1893, under Dr. Pye-Smith, when she remained in the hospital until July 2nd, 1893. Mitral stenosis, tricuspid regurgitation, tricuspid steuosis and enlarged liver were diagnosed. On June 12th, 1893, *Dr. Pye-Smith heard a diastolic bruit in the fourth left space with reduplication of the second sound in the third left space.* On June 23rd, 1893, there was a systolic bruit at the ensiform cartilage, a presystolic bruit at the apex, *and a faint short diastolic bruit in the third left space coming*

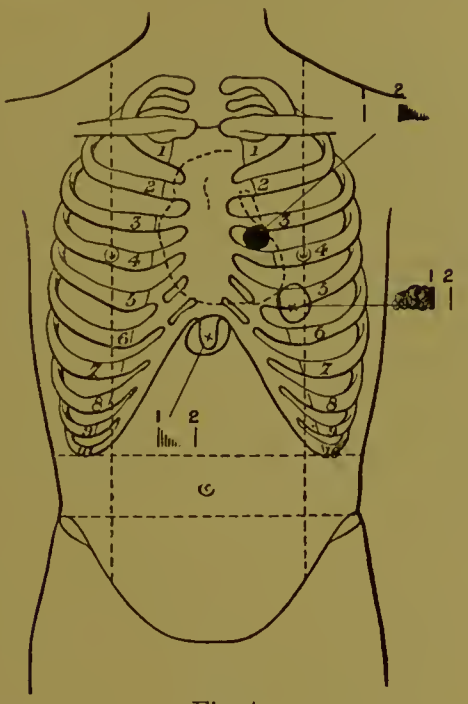

Fig. 4.

immediately after a very accentuated second sound. (P.S. 103, 1893. Clerk, R. Henderson.)

She was readmitted on July 16th, 1894, and remained in until July 25th, 1894, under Dr. Shaw. The bruits were then presystolic (no thrill) and systolic at the apex, systolic at the ensiform cartilage, while there was a reduplicated second sound heard to the right of the sternum, aud an accentuated second sound in the pulmonary area. The liver reached 5 centimetres below the costal margin. The diagnosis was acute supervening on chronic bronchitis, mitral stenosis and tricuspid regurgitation. (C. 336, 1994. Clinical, W. E. Robinson.)

She was admitted again, October 27th, 1894, under Dr. Taylor, with signs of extreme backward pressure, and venesection was performed, which greatly relieved her. She had a long presystolic bruit at the apex, with no recognisable second sound, and there was a squeaking systolic bruit over the ensiform cartilage. Her fingers were clubbed, the liver was three fingers' breadth below the ribs, and her feet and ankles were œdematous. The diagnosis made was mitral stenosis, tricuspid regurgitation and acute bronchitis. She went out again February 20th, 1895. (C. 1, 1895. Clinical, A. Salter.)

Condition on admission, 15th May, 1895. Temperature 99°, pulse 120, respiration 56.—There was some œdema of the legs and ankles, and some ascites. Pulse regular, low tension; no pulsation of the veins of the neck. The cardiac impulse was very diffuse, and was felt in the fifth and sixth spaces, extending in the sixth space as far as five centimetres outside the nipple-line. There was no thrill. Some epigastric pulsation was noticed. The cardiac dulness extended above to the fourth rib, externally to the nipple-line, and internally

to the mid-sternal line. At the impulse there was a loud, rough presystolic murmur running up to an accentuated first sound, followed by a soft systolic murmur, with no recognizable second sound. Traced out into the axilla, the first sound was replaced by a systolic bruit. Over the lower end of the sternum and in the epigastrium, a softer blowing systolic murmur was heard, followed by a second sound. The murmur could be traced to within 4 centimetres of the right nipple. In the pulmonary and aortic areas an early diastolic murmur was heard, not distinguishable to the right of the sternum (?). Numerous râles and rhonchi were heard all over the chest. The liver extended 5 centimetres below the costal margin, and the urine contained a trace of albumen. On May 17th, the early diastolic murmur in the pulmonary area had disappeared; but it was just audible again on May 18th. On May 18th it was again not heard. On May 23rd Dr. Goodhart heard a systolic bruit in the pulmonary area. On May 29th the early diastolic murmur in the third left space near the sternum was again heard (J. H. B.). It was noted again on June 5th, but was not mentioned after. Patient left the hospital on July 17th, 1895.

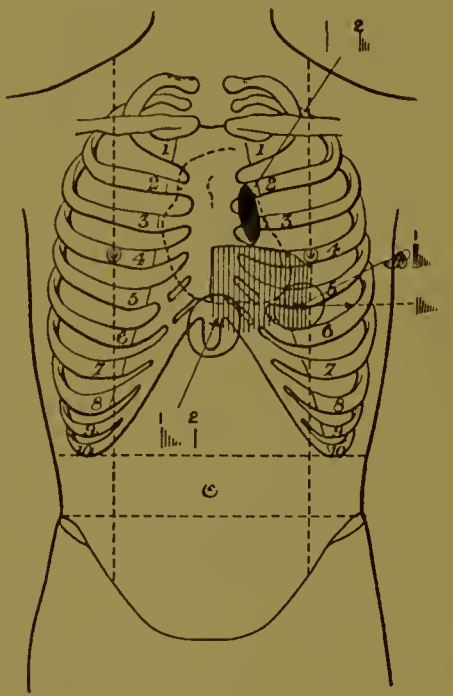

Fig. 5.

She was readmitted September 20th, 1895, under Dr. Pitt (C. 493, 1895. Clinicals, A. V. Clarke and E. Erskine Henderson).

Condition on admission.–Pulse 100, irregular and very feeble. Much cyanosis. Veins of neck pulsating, considerable œdema of the legs and feet. Cardiac dulness was the same as on May 15th, 1895. The impulse was diffuse in the fifth space one inch internal to the left nipple. No thrill. Systolic and presystolic murmurs at the apex, no second sound. Systolic murmur audible at the base. There were râles and slightly impaired resonance at both bases. The

liver was soft, pulsating and tender, and extended 5 centimetres below the costal margin. The urine contained a trace of albumen. On October 2nd the bruits were a presystolic at the apex where no second sound was audible, and a systolic bruit over the lower end of the sternum where the second sound was heard. A systolic bruit was heard at the base and thought to be the tricuspid bruit transmitted. On November 12th she had another attack of bronchitis and died on November 18th, 1895.

Autopsy 459, 1895, by J. H. Bryant.—The body was fairly well nourished and the face was cyanosed. The pleura covering the right lower lobe of the lung was very much thickened, and there was evidence of old pleurisy over the left lower lobe and also over the posterior surface of the upper lobe. The bronchial glands were enlarged and the lungs were tough and of a reddish-brown colour. There was very little œdema and no infarcts. The trachea and bronchi were congested. There was no pericarditis. The heart weighed 350 grammes and the right side was hypertrophied and a little dilated. The wall of the right ventricle measured 9·5 millimetres in thickness and the muscle was good. The right auricle was a little dilated. The tricuspid valve measured 10·4 centimetres and the pulmonary orifice 7·62 centimetres. The tricuspid valve was slightly thickened. The left ventricle was not hypertrophied, but the left auricle was both hypertrophied and dilated. There were a few recent vegetations on the auricular edge of the mitral valve, which was 3·17 centimetres in circumference and was much thickened and calcareous. The chordæ tendinæ were short, thickened and adhering to each other. The aortic valves were healthy. *The pulmonary artery was thickened and dilated; it was almost as thick as the aorta. 2·5 centimetres above the pulmonary valves it measured 8·25 centimetres in circumference when laid open. The two main branches were thickened and atheromatous, and the small branches in the lungs were thickened, atheromatous and dilated, and stood out prominently from the cut surface of the lung, with open lumina.* The stomach was congested, and there were a few submucous petechiæ. The duodenum was congested. There was no ascites. The liver was soft and weighed 1304 grammes; it was not typically nutmeg. The spleen was hard and weighed 170 grammes. The kidneys weighed 340 grammes; they were very hard and congested and the cortex was scarred in places and the capsule thickened.

Case 4.—Eliza W., æt. 32, admitted June 9th, 1894, under the care of Dr. Pitt (clerk, C. L. Hopkins), for anasarca and dyspnœa. Her father had twice had rheumatism, and one of her brothers had also suffered from rheumatism. In 1889, she had an attack of rheumatism, and another in July, 1893. At this time the cardiac impulse was found to be just inside the left nipple-line in the fifth space. The cardiac dulness was normal. There was a harsh presystolic bruit at the apex, and the aortic and pulmonary sounds were accentuated. A diagnosis of acute rheumatism with mitral stenosis was made. She had been married fourteen years, and had had five children. On December 11th, 1893, she was admitted for marked dropsy. In addition to the mitral bruit, a tricuspid regurgitant murmur was heard. There was no bruit at the base, but the second sound was reduplicated. She went out relieved at the end of the month.

When admitted on June 9th, 1894, she was in a collapsed condition, and was very dyspnœic and cyanosed. The pulse could not be felt at the wrist.

There was very extensive œdema of the legs, and there was ascites. The heart was beating 132 to the minute. On the following day the apex beat was found to be diffuse and irregular, and was in the sixth space. Four centimetres outside the nipple-line there was marked epigastric pulsation. The cardiac dulness commenced above at the third rib, to the left it was limited by a line 13 millimetres to the right of the right border of the sternum, and outwards by a line 25·4 millimetres to the left of the left nipple-line. There was a presystolic thrill at the apex. A rough localised presystolic bruit was heard at the cardiac impulse, followed by a blowing systolic murmur, which could be traced outwards. In the aortic area the sounds were faint. The urine was 1020, and contained a trace of albumen. There was marked ascites, and in consequence the liver could not be felt. On the 11th she was better. She was so dyspnœic, however, that she could not lie down. On the 13th she was much about the same in the morning; but about 7 p.m. she was very much worse, becoming gradually cyanosed and breathless. The hands and forehead became cold. The heart beat was rapid, fluttering, irregular and uncountable. The bases of the lungs were resonant. Dr. Pitt suggested that the sudden change was due to pulmonary thrombosis. At 5 a.m. she became worse; an injection of Liq. Strychnine mv. was given, and oxygen was administered. Towards evening she became better. She again became worse on the 14th, and died at 12.15 p.m.

Post-mortem, 234, 1894.—The *post-mortem* was made by Dr. Perry fourteen hours after her death. There was much œdema of the lower extremities. The right pleural cavity contained 426 cubic centimetres, and the left 341 cubic centimetres of serous fluid. There were filamentous adhesions on the right side. The lungs were in a condition of brown induration, and there were definite hæmorrhages into the substance at the lower part of both lower lobes. The heart weighed 482 grammes. Both sides were large and dilated. *On the right side the pulmonary valves measured 11·4 centemetres in circumference, and therefore there was doubtless pulmonary regurgitation. In the artery were atheromatous patches, and in the left branch of it a small piece of ante-mortem clot*, which, however, did not at all occlude the lumen of the vessel to any important extent. The tricuspid orifice was 15 centimetres in circumference. On the left side the mitral orifice just admitted the index finger, and the aortic valves, which were competent to all appearances, measured 6·35 centimetres in circumference. The aorta was therefore small. There was ante-mortem thrombus in both the right and left appendices auriculæ. There was also an ante-mortem thrombus in the abdominal aorta, just above its division into the common iliacs. The left ventricle was very slightly enlarged. The œsophagus, stomach and intestines were a good deal congested. The liver weighed 1673 grammes, and was nutmegged. The spleen weighed 170 grammes; it was hard and scarred, and showed an organising infarct. The kidneys weighed 397 grammes; they were hard and scarred from infarction.

Case 5.—Fred. J., æt. 20, photographer, was admitted under the care of Dr. Washbourn, on December 8th, 1897, for shortness of breath. Five years ago he had some pain and swelling in his joints. He first noticed swelling of his hands and arms at the end of the bicycle racing season; about fourteen months before, this was followed by shortness of breath, and he had to give up bicycling.

Condition on admission.—Pulse very rapid and irregular. The cardiac impulse was very diffused and extended from the nipple line in the fifth space to the border of the sternum. There was no thrill. The cardiac dulness commenced above at the third rib, extending outwards to the left nipple line and inwards to the mid sternal line. There was a systolic bruit heard at the apex which could be traced inwards to the sternum and outwards to the anterior axillary line. The sounds at the base appeared to be normal. On December 11th, the pulmonary second sound was noted as being accentuated. On the 15th, he complained of tonsillitis. On the 16th, a pleuritic rub was heard at the left base behind. On the 21st, mitral and tricuspid regurgitant bruits were audible. On January 4th, pleuritic effusion was noted. On January 7th, purpuric spots were noticed on the thighs and abdomen. The urine was 1022 and contained blood and albumen. On January 10th, an apical diastolic bruit was heard. He was much worse; the purpuric rash had spread, and he died at 6.25 a.m. on the 11th.

Pulmonary regurgitation was not diagnosed, nor in fact was mitral stenosis. He was thought to be suffering from mitral and tricuspid regurgitation.

Post-mortem 19, 1898.—The post-mortem examination was made seven hours after death. Rigor mortis was well marked. There was considerable œdema of the legs. Numerous petechiæ and ecchymoses were seen over the abdomen, back, and upper part of the thighs. The thyroid was uniformly enlarged. Each pleural cavity contained about a litre of clear, pale serous fluid. There was recent pleurisy on the edge of the left lung. The pleuræ were thickened and adherent over the base and posterior surface of the left lower lobe. Both lower lobes were a good deal compressed Both lungs were tough and in a state of red induration. *The branches of the pulmonary arteries in the lungs were thickened and atheromatous, standing out prominently with open lumina from the cut surface of the lung.* There was no pericarditis. The heart weighed 596 grammes. There was a slight amount of fat on the anterior surface of the right ventricle. The coronary arteries were dilated. The right ventricle measured 11·43 centimetres across at the base, its wall was hypertrophied and the cavity was dilated. The tricuspid orifice measured 10·16 centimetres, and the valve was a little thickened. The right auricle was dilated; the pulmonary veins were normal. *The pulmonary artery was dilated.* There was some ante-mortem blood-clot in the appendix of the left auricle. The left auricle was hypertrophied and dilated. The endocardium was thickened. The left ventricle was dilated. The mitral valve was thickened and calcareous and its orifice was much narrowed and measured five centimetres in circumference. The chordæ tendiniæ were thickened and shortened. The aortic valves were slightly thickened and adherent, but appeared to be competent. The stomach was contracted; its mucous membrane was congested. There were several cubic centimetres of clear serous fluid in the peritoneal cavity. The liver weighed 1813 grammes; on section it presented a typical nutmeg appearance. The spleen weighed 368·5 grammes; it was very hard; the suprarenal capsules were normal. The kidneys weighed 411 grammes; the capsules were a little thickened; both kidneys were extremely hard and congested. There was no sign of embolism; there was no macroscopic appearance of nephritis. The hæmaturia was probably due to congestion.

Case 6.—Henry W., æt. 22, a hawker, was admitted on January 19th, 1894, under the care of Dr. Taylor (clinical clerk, R. G. Dellbruck) for palpitation of the heart and pain in the chest. He had previously been in the hospital on two occasions, first under Dr. Hale White and then under Dr. Washbourn. A diaguosis of mitral stenosis aud regurgitation was made on both occasions. His father died of heart disease and his mother suffered from palpitation. When eight years of age he suffered from scarlet fever. No history of rheumatism or chorea could be obtained. In the summer of 1892 he began to have attacks of cardiac syncope. In the winters of 1892 and 1893 he had bronchitis. For the six months previous to his admission he had had dropsy.

Condition on admission.—Pulse 92, full and regular. The left side of the chest near the sternum was a little more prominent than the corresponding part on the right side. The impulse was diffuse and was in the fifth left space, just to the left of the left uipple line. The cardiac dulness commenced above at the fourth rib, exteuded inwards to the right border of the steruum and outwards to a line 2·5 centimetres exterual to the left nipple line. Iu the sixth space, about 4 centimetres outside the impulse a loud, rumbling presystolic murmur runuing up to a loud first sound, which was immediately followed by a systolic murmur, was heard. At the point of the cardiac impulse a loud systolic murmur was heard. The second sounds at the base were accentuated. There were no basal bruits. There was no marked pulsation of the carotids or other arteries. On January 27th, he had two sharp attacks of palpitation. On February 9th, on auscultating just outside the uipple in the fifth left space the sounds had a different character on light and firm pressure of the stethoscope. On light pressure a booming sound was heard which appeared to occupy the whole rhythm of the heart and was heard alone during diastole, a blowing murmur being heard during systole. On firm pressure a triple sound was heard, the first sound appearing to be reduplicated, though the heart was beating too rapidly to be sure that it was the first sound that was reduplicated. The booming sound disappeared, but the blowing systolic bruit retained its character. On February 23rd Dr. Taylor writes:—"Systolic murmurs as above described, marked systolic thrill over impulse, and nearly to sternum. Diastolic murmurs as before in impulse and in axilla." Pulsating liver. He lies drowsy, pallid, dusky, with rapid breathing—36. Pulse small, regular, feeble; not specially indicative of any valvular disease. On the 24th he became slightly jaundiced, and was much worse. He died at six p.m.

Post-mortem (by J. H. Bryant).—Performed twenty hours after death. Rigor mortis was slight. There was marked hypostasis of the back. There was no recent pleurisy, but there were old fibrous filamentous adhesions over the posterior surfaces of both lungs. The lungs were tough and hard, in a condition of marked red splenization. *On section, the pulmonary arteries stood out very prominently from the cut surface; they were dilated, very much thicker than normal, showing marked atheromatous changes, appearing in fact almost as thick as similar-sized bronchial tubes.* There was no pericarditis. The heart weighed 510 grammes. On opening the pericardial sac and looking at the anterior surface of the heart it appeared to be almost entirely made up of the right ventricle, the left ventricle only just being visible, the right ventricle at least consisted of five-sixths, the left only one-

sixth of the two ventricles together. The wall of the right ventricle measured two centimetres in thickness. The right auricle was also hypertrophied and very much dilated. *The pulmonary orifice measured seven centimetres, just above the orifice the artery measured 8·8 centimetres and had the appearance of being considerably dilated, and was much larger than the aorta.* The tricuspid orifice measured *14 centimetres.* The left ventricle was very small and its wall measured just under 13 millimetres in thickness. The curtains of the mitral valve were very much thickened and extremely hard. The tendinous cords were short and very thick. The mitral orifice would only admit the top of the little finger and measured 37 millimetres in circumference. The endocardium of the left auricle was thickened. The aortic valves were healthy. As above mentioned, the pulmonary artery was dilated just above the valves, and atheroma was marked in the branches commencing in the two first divisions.

Case 7.—Julia D., æt. 27, was admitted on October 23rd, 1890, under the care of Dr. Hale White (clinical clerk, A. H. Trevor), for her third attack of acute rheumatism. Seven years before she was under the care of Dr. Taylor for mitral stenosis, and nine years before this she was also in the hospital for acute rheumatism and pericarditis. On this occasion a systolic apical murmur was also noted. Pain and swelling of her right elbow appeared on October 17th, and kept her from her work; on the two following days other joints were affected, and she kept her bed until the 23rd, when she came to the hospital, and was admitted.

Condition on admission.—Pale and emaciated, lips livid; the pulse was weak, rapid and compressible. The cardiac impulse was seen and felt in the fifth left intercostal space in the nipple line. No thrill could be felt. The cardiac dulness was increased to the right border of the sternum, above to the third rib, and to the left to the left nipple line. On auscultation, at the point of the cardiac impulse a loud presystolic murmur was heard running up to and continuous with a loud first sound. At the base, a blowing systolic murmur was heard, chiefly on the right side of the sternum. The second sound was accentuated in the second left intercostal space. Rhonchi and râles were heard at both bases. The tongue was covered with a thick brown fur. The liver was felt below the right costal margin. The urine was 1026; it did not contain albumen or sugar. On October 29th, Dr. Hale White heard a pleuritic rub in the left axillary region. On November 5th, a systolic bruit was heard at the apex, which was traceable outwards into the axilla. On November 7th, the following report was made:—"Patient's pulse was very slow yesterday afternoon (50) (? due to digitalis). This morning it is 56, and on auscultation it corresponds to the cardiac beats. A systolic murmur, and also a presystolic murmur can be heard at the apex, but 25 millimetres outside the apex nothing can be heard but a very sharp loud first sound, and occasionally a very feeble second sound. The systolic murmur can be heard all over the base, but is clearest at the left edge of the lower part of the sternum. The second sound is very much accentuated in the second left space, where also a systolic and *a faint diastolic murmur can be heard.* The first sound can be plainly heard by listening at the patient's back through the flannel vest." November 9th.—A mid-diastolic bruit was heard at the apex, and a diastolic thrill was felt. November 10th.—The patient became very dyspnœic, and died at 10.15 p.m.

The *post-mortem* was performed by Dr. E. W. Goodall, twenty-nine hours after her death. The body was pale and thin. There was slight œdema of the legs. The pleura was thickened over the right upper lobe. There was recent pleurisy involving the rest of the right lung and the whole of the left. The lungs were in a condition of brown induration. There were a few recent infarcts, the largest being in the left lower lobe behind. The pericardium was universally adherent, but could be stripped off in front. The heart weighed 510 grammes. There was some ante-mortem clot in the appendix of the right auricle, and amongst the papillary muscles of the right ventricle. The right ventricle and auricle were both dilated and hypertrophied. The tricuspid valve was much shortened and thickened, the circumference of its orifice was 12·7 centimetres. *The pulmonary orifice measured 7·6 centimetres. The pulmonary artery was dilated, and was much larger than the aorta. The intima was thickened, and the larger branches showed marked atheromatous changes.* The right ventricle showed several fibroid patches in the septum ventriculorum and at the apex. The left auricle was considerably dilated and hypertrophied, and the endocardium was much thickened. The left ventricle was slightly dilated and hypertrophied. The mitral orifice was much narrowed and the valve button-hole. The orifice would only admit the tip of the little finger. The valve segments were rigid, very short and thick. The chordæ tendiniæ were glued together and short, and there was marked fibroid change in the upper parts of the papillary muscles. The aortic orifice measured 5·6 centimetres. The valves were quite competent, although the valve segments themselves were certainly thickened and also the corpora-arantii. Liver, 1474 grammes; marked nutmeg appearance on section. Spleen, 142 grammes, softer than usual in a mitral case. The capsule was thickened. Kidneys, 340 grammes; very firm; the remains of a few old infarcts were seen here and there in each organ.

Case 8.—Eliza S., æt. 25, admitted on July 1st, 1888, under the care of Dr. Perry (clinical clerk P. Paget) for dyspnœa. She was first admitted on January 13th, 1888, for mitral stenosis, tricuspid regurgitation, bronchitis and pregnancy, and went out on February 22nd relieved. She was again admitted on March 7th, and was discharged relieved on April 13th. There was a history of rheumatic fever in 1882.

Condition on admission.—She was pale and dyspnœic, but was not cyanosed. The pulse was 90, irregular, intermittent and compressible. The cardiac impulse was diffuse and could be seen in the fifth, sixth, and seventh spaces, in and outside the left nipple line. A well marked thrill could be felt at the apex. The cardiac dulness was increased a little in all directions. On auscultation, a murmur of maximum intensity at the apex was heard, and it could be traced outwards into the axilla: it was of a sharp blowing character and occurred with the first sound. There was no presystolic bruit. There was marked œdema of the legs and back and many purpuric spots were seen on the legs. The urine was 1020; it did not contain any albumen. She was put on ten minim doses of Tinct. Digitalis. On the 22nd, the pulse was better. It was noticed that she was slightly cyanosed. On the 23rd, she was more cyanosed and was very orthopnœic, and a well marked presystolic bruit was heard. The chest was explored for fluid, but none was found. On the 24th, as she was restless, an injection of Morphia gr. ⅓ was given, and after this

she vomited and her pupils became contracted; at 2.15 p.m. she was very cold and almost pulseless; she died at 2.15 p.m.

Post-mortem, 255, 1888.—The post-mortem was performed fifteen hours after death. The body was much cyanosed, there was marked œdema of the legs, and there were a few purpuric spots on the legs. The pleura was thickened and adherent over the posterior surface of the right lung. The lungs were in a condition of brown induration. There was a small infarct in the lower and outer part of the left upper lobe. The pericardial sac contained 313 cubic centimetres of brownish serous fluid. The pericardium covering the heart was thickened. The heart weighed 397 grammes, and it was broadened by the increase in the size of the right ventricle. The left auricle was much hypertrophied and dilated and its walls were tough and leathery. The mitral valve was converted into a calcareous ring, just admitting the end of the index finger; there were a few vegetations on the ring. The left ventricle was slightly hypertrophied; it was not dilated. The edges of the aortic valves were thickened and rolled. It seemed as though there must have been some regurgitation, but no murmur was ever heard, and the left ventricle was not much enlarged. The right auricle and ventricle were dilated and hypertrophied. The tricuspid valve had thickened edges and allowed regurgitation. It measured 10 centimetres across. *The pulmonary artery was much larger than the aorta, was much thicker, and had atheromatous patches in it. It measured 7·6 centimetres across against 5·7 centimetres of the aorta.* The *liver* weighed 1758 grammes, it was nutmegged and fatty in appearance. The spleen weighed 255 grammes, it was hard. The kidneys weighed 283 grammes. There was some scarring from infarcts.

Case 9.—David L., was admitted on March 2nd, 1887, under the care of Dr. Taylor (clinical clerk R. G. Hicks) for dyspnœa. He had twice suffered from rheumatic fever; first of all in 1848 and again in 1871. About Christmas he first noticed a little difficulty in breathing and since that time it gradually became worse. About a fortnight before his admission to the hospital he experienced one night a good deal of difficulty in getting off his stockings, and found that his legs and ankles were much swollen.

Condition on admission.—He was a fairly healthy-looking man; his eyelids were a little puffed and there was marked œdema of the legs and feet. The pulse was 98, full, soft and regular. The cardiac impulse was in the sixth space 2·5 centimetres outside the nipple line. The impulse was rather forcible. There was distinct systolic retraction in the epigastrium. A loud systolic bruit was heard at the apex and it could be traced outwards into the left axilla, but it could not be heard behind at the angle of the scapula. The respirations were 24; râles and rhonchi were heard at both bases. The liver was enlarged and tender and its edge could be felt at the level of the umbilicus. The urine was 1017, and it contained albumen. On March 3rd, there was no albumen in the urine, and the œdema was less. On March 5th, he had a choking fit, he was asleep at the time; he suddenly started up in bed with a feeling of suffocation as if something was rising in his throat. The pupils were unequal, the right being much smaller than the left. On March 11th, there was dulness at both bases, and he was found to be expectorating blood-stained frothy mucus. On March 15th, a well-marked pleuritic rub was heard in the right axilla. On March 17th, there were signs

of pleuritic effusion on the right side. On March 20th, aspiration was performed and 227 cubic centimetres of clear serous fluid were withdrawn. On April 3rd, he had several attacks of dyspnœa. On April 7th, he felt worse. He was still bringing up the blood-stained sputum. On April 9th, Cheyne-Stokes respiration was noted. On April 14th, he was much weaker, thinner and paler. He gradually became worse and died at 1 a.m. on the 15th.

Post-mortem 96, 1887.—The post-mortem was made by Dr. Pitt fourteen hours after death. The body was rather wasted; there was very little œdema. There was about half a litre of pus in the right pleural cavity, and there was a corresponding amount of compression of the right lower lobe. The pericardium was universally adherent but was not much thicker than normal; over the portion adherent to the right ventricle was a small, thick calcareous mass, for which no cause was obvious. The myocardium was fatty and mottled in colour on both sides. Both ventricles were dilated, especially the right; the left auricle was greatly dilated. The left ventricular wall measured 16 millimetres. There was chronic thickening of the endocardium on the left side. The mitral valve was stenosed; it measured 7·6 centimetres. The aorta was dilated and the valves were thickened and doubtfully competent. The tricuspid valve was thickened and it measured 14·3 centimetres. *The pulmonary artery looked very large, the valves measured 8·9 centimetres and were normal in appearance. The pulmonary branches were greatly dilated and the coat showed irregular cracks in the intima, which were blood stained.* These appeared to be due to over distension; there was no atheroma. There were numerous calcareous and fibroid patches in the aorta, especially in the first part. The liver weighed 1758 grammes; it was slightly nutmegged. The spleen weighed 255 grammes; it was tough. The kidneys weighed 312 grammes; they were tough and congested.

Case 10.—N. H., an unmarried woman, æt. 23, came to my Out-patients for dyspnœa on October 29th, 1900. She had been troubled with shortness of breath for several years, getting it if she walked quickly and when she went upstairs. Five or six years ago she says that one day when coughing she burst a blood-vessel and brought up a quantity of blood. She states also that she has frequently brought up blood since, especially after exertion of any kind. Her menses are irregular. In June last she suffered from "quinsy." She gives no history of either rheumatism or scarlet fever. She says she has not lost flesh.

Condition.—Pulse 120, temperature 98·4°. There was marked dyspnœa, the alæ nasæ acting freely. There was no œdema of the legs and no ascites. The pulse was 120, a little irregular, and easily compressible. The cardiac impulse was seen in the sixth space about two centimetres outside the left nipple line; it was forcible in character and was also diffused. There was some epigastric pulsation. There was also a little bulging of the chest in the precordial area. A well-marked presystolic thrill was felt over the cardiac impulse. The cardiac dulness was increased (*vide* diagram) At the cardiac impulse a loud rumbling presystolic murmur was heard running up to and being continuous with a loud accentuated and prolonged first sound, which was followed by a blowing systolic murmur. *In the second and third spaces midway between the left border of the sternum and the left nipple line a soft, blowing, early diastolic murmur was heard.* Tho

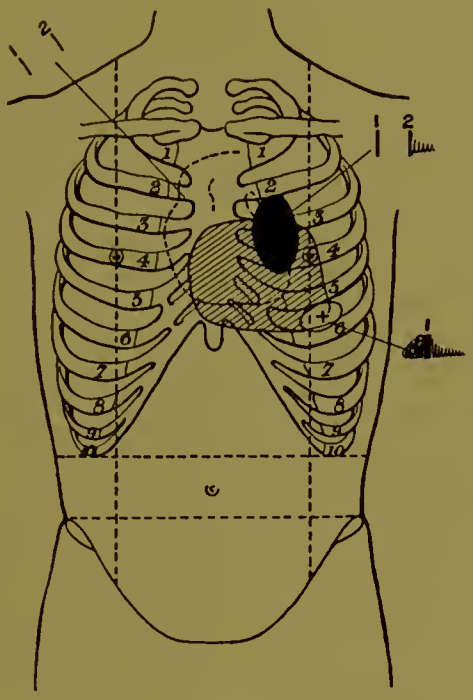

Fig. 6.

aortic second sound was sharp, clear and accentuated. She has been seen on several occasions and the diastolic bruit has always been present in the position described.

Case 11.—Susan H. M., æt. 14, under the care of Dr. Pye-Smith (clerk B. W. Moss), admitted February 17th, 1898, for precordial pain and cough. No family history of rheumatism. She has never been a strong child; four years ago she was admitted under Dr. Pye-Smith for chorea, and two years ago she was under Dr. Taylor, for rheumatism. When under Dr. Pye-Smith a diagnosis of mitral stenosis and regurgitation was made, the bruits heard being systolic and mid-diastolic in rhythm. When under Dr. Taylor, presystolic and systolic bruits were heard in the mitral area and a systolic bruit in the pulmonary area. For the last twelve months she has been losing flesh, and her appetite has been very capricious, and for ten months previous to admission her cough has been very bad, and she has been short of breath. She has also suffered from pain and swelling at the back of her knees. There has been no swelling of her feet or ankles; no hæmoptysis or hæmatemesis.

Condition on admission.—Temperature 100°, pulse 100. She was a pale, thin, weakly looking child and did not appear to be in pain. *Circulatory system.*—Pulse 100, regular, weak and compressible. There was slight bulging of the chest in the precordial area. The impulse was visible in the fifth space, it was diffused in character and extended outwards about 2 centimetres external to the left nipple line. A presystolic thrill was felt over the mitral area. The cardiac dulness commenced above at the third left costal cartilage and extended below to the sixth; to the right it was limited

by the left border of the sternum, and to the left by a line drawn from the third rib through the nipple to the impulse. At the apex a loud, rough, rumbling presystolic bruit was heard, running up to a loud, sharp first sound, which was immediately followed by a harsh blowing systolic murmur, which latter bruit could be traced outwards into the left axilla. The presystolic

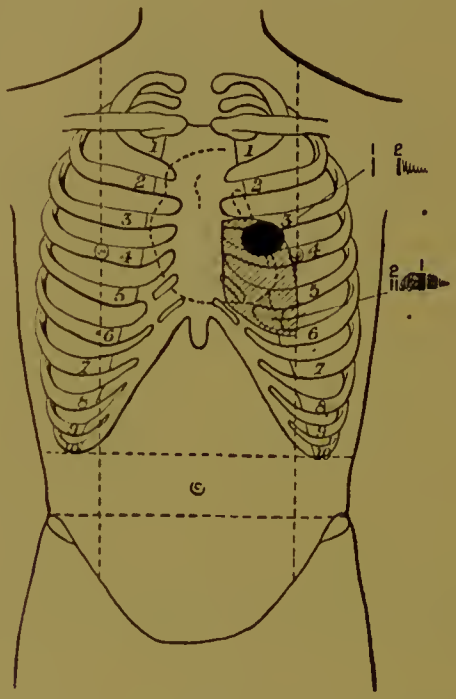

Fig. 7.

bruit followed immediately after a double second sound. *In the third left space, about 2 centimetres from the left border of the sternum, and 1·5 centimetres internal to the left nipple line, a faint, soft, blowing early diastolic murmur could be heard.* The aortic second sound was clear and distinct and there was no visible pulsation of the carotid arteries. No abnormal respiratory sounds. No ascites. Urine normal.

The patient left the hospital on March 2nd. The bruits at the apex altered from time to time, the presystolic being replaced by a mid diastolic bruit. The early diastolic murmur in the third left space, however, persisted, but it was not quite so well marked.

Case 12.—Priscilla C., æt. 39, under the care of Dr. Shaw (clerk A. W. Talbot), Mary 19, and admitted on February 24th, 1898, for cough, dyspnœa and precordial pain. There was no family history of rheumatism. Her father suffered from gout and died at the age of eighty-one. She was married in 1888; she has no children. She has a cough every winter and expectorates a large quantity of phlegm. Two years ago, when hurrying to business, she had an attack of hæmoptysis. About November, 1897, she noticed dyspnœa on exertion. About a month previous to admission she began to suffer from precordial pain; fourteen days later the pain became

much worse, and it was so intense that it made her cry out, and as it gradually became still worse she came up to the hospital and was admitted on February 24th.

Condition on admission.—Temperature 97·6°, pulse 136, respiration 44; much cyanosed, veins in the neck pulsating. February 28th. *Circulatory system.*— Pulse 136, irregular, intermittent and compressible. Cardiac impulse visible in the fifth left space 2·5 centimetres outside the left nipple line, diffused and irregular; no thrill felt. Cardiac dulness is limited above by the third rib, to the right by the mid sternal line, below by the fifth space, and to the left by a line drawn downwards and outwards starting above just inside the left nipple line. On auscultation at the cardiac impulse the first sound was very loud, sharp, and slapping in character; it was preceded by a short, loud, rumbling presystolic murmur. The second sound in this position was loud and was followed by a soft, early, diastolic murmur. Just outside the impulse the first sound was loud and sharp in character and the early diastolic murmur was also audible. *In the second and third left spaces midway between the left border of the sternum and the left nipple line a soft, early, diastolic murmur, which appeared to be different in its character to the above mentioned early diastolic murmur audible at the impulse, was heard. No bruits were heard in the aortic area.* A few râles were heard at the bases of the lungs behind. On March 2nd, the presystolic murmur could not be heard. The early diastolic murmur in the pulmonary area was, if anything, more distinct.

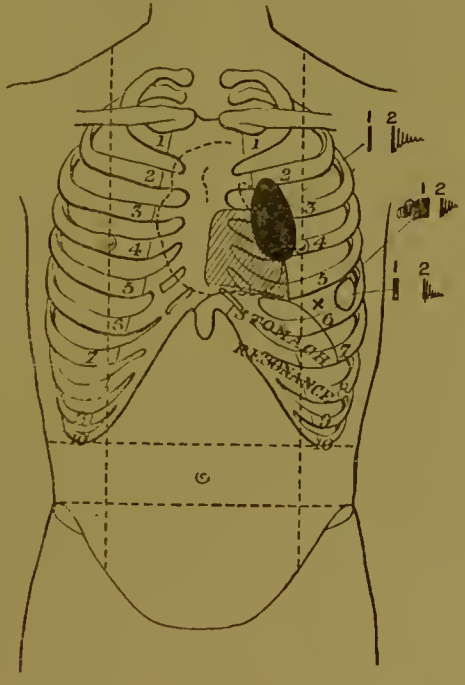

Fig. 8.

Case 13.—Emily K., æt. 8, under the care of Dr. Goodhart (clerk H. E. C. Fox), admitted October 9th, 1897, for pain in the chest and shoulders. No family history of rheumatism or chorea. She has always been a delicate child, and when quite young suffered from an attack of congestion of the lungs following measles. Six years ago she had a bad attack of scarlet fever, which left her in a very weak state; since then she has suffered at times from fainting fits. Six weeks before admission the mother first noticed symptoms of chorea, viz., involuntary twitchings of the muscles of the hands and face.

Condition on admission.—Pulse 120, respiration 36, temperature 101·2°. No signs of chorea. The cardiac impulse was visible in the fourth space in the nipple line. A presystolic thrill was felt over the mitral area. For cardiac dulness *vide* diagram. On auscultation, a presystolic bruit was heard,

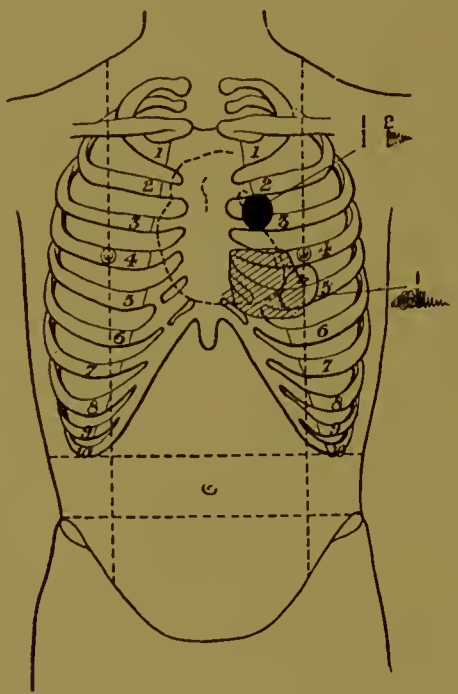

Fig. 9.

running up to a loud accentuated first sound, which was immediately followed by a loud blowing systolic murmur, traceable outwards into the left axilla. On October 21st, a loud and accentuated second sound *followed by a soft, blowing, early diastolic murmur was heard over the second left space just outside the left border of the sternum. The aortic second sound was sharp and clear and no bruit could be heard accompanying or following it.* On November 4th, the second sound in the pulmonary area was loud and slapping in character and there was immediately following it a well-marked blowing diastolic murmur. On January 3rd, 1897, she was discharged. She was very much better and the pulmonary diastolic bruit was not audible,

Case 14.—M. H., æt. 12, a schoolgirl, was admitted 23rd February, 1897, under the care of Dr. Taylor (clinical clerks E. B. Dowsett and D. Munro) for dyspnœa and swelling of feet. There was no history of any previous illness, no scarlet fever, no rheumatism and no chorea, though the patient had always been weakly. The family history was good. She had generally had a winter cough and sometimes had had pain in her shoulder. The present illness had commenced two years ago, after a fall from a ladder, when she injured her left knee. She had limped since the accident, had lost flesh and had complained of shortness of breath, palpitation and pain at her heart. About the 3rd February she had become much worse had an aggravating dry cough, her ankles had swollen and she had been obliged to sit up at night for breath. Latterly the pain at her heart had been worse, and her face had become purplish. There had been no vomiting and no hæmoptysis.

Condition on admission.—Temperature 98°, pulse 100, respiration 24. She was thin and anæmic. Both knees were large but showed no signs of injury. The venules on the face were injected and prominent. *Circulatory system.*—Pulse regular, full, dicrotic, easily compressible. There was marked bulging of the precordia. The cardiac impulse was very diffuse, most marked in the sixth space 2·5 centimetres external to the nipple, but also seen in the seventh space 4 centimetres external to the nipple. Some sucking in of the intercostal spaces during systole was noticed. The cardiac dulness was limited above by the second left space, internally by the mid-line of the sternum, and it extended outwards to a point two centimetres external to the nipple, but there was partial dulness over a larger area (*vide* diagram). A well marked presystolic thrill

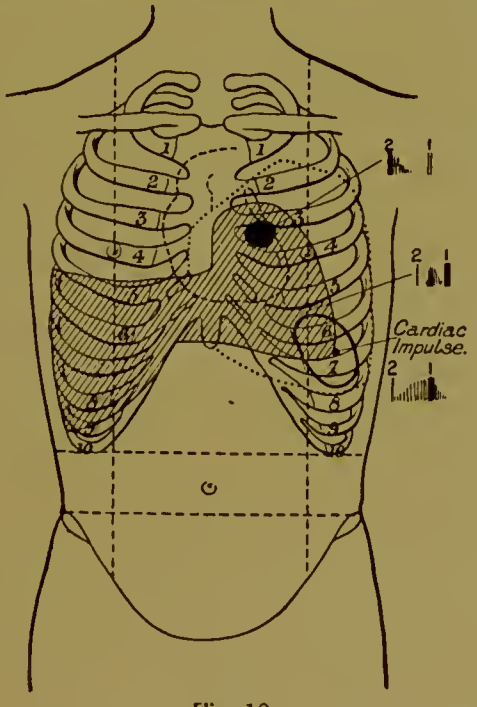

Fig. 10.

was felt over the cardiac impulse. A loud rumbling presystolic bruit with a loud first sound, was heard at the apex, followed by a faint

blowing systolic murmur traceable outwards. *In the third left space, near the sternum, the first sound was reduplicated, the second sound was loud and was followed by a faint blowing diastolic murmur.* Respiration was laboured in the supine position, but was easy and regular when the patient was propped up with pillows. There was a troublesome cough but no expectoration. There was some dulness at the left base as high as the angle of the scapula and some bronchophony along the vertebral border of the left scapula. Loud rhonchi were heard over the upper half of the right chest and some very fine crackling râles were audible on both sides on deep inspiration. The *urine* was acid, sp. gr. 1018, and contained a trace of albumen. The *abdomen* appeared normal. Mitral stenosis and regurgitation was diagnosed. On March 1st, a soft systolic bruit was heard over the ensiform cartilage. The reduplication of the first sound in the third left space was more marked. On March 2nd, the diastolic murmur in the third left space was more marked but very localised. On March 4th, a soft blowing systolic murmur was heard in the second right space. On March 12th, the diastolic murmur in the third left space disappeared and was not heard again during the remainder of patient's stay in the hospital.

Patient continued to improve, and when she was discharged on March 23rd, the systolic bruit at the apex was loud and traceable into the axilla and behind. There was also a well-marked presystolic bruit (? at apex). The pulse was 120, regular and soft, and not nearly so dicrotic in character. There was still some dulness at the left base over which crackling râles were heard, and a loud rhonchus was audible at the left apex posteriorly.

She was readmitted April 1st, for herpes zoster of the right buttock and outer side and front of right thigh. The condition of the heart was about the same, but no diastolic bruit was heard in the third left space. She left the hospital again on April 17th.

Case 15.—J. A., æt. 15 years, schoolboy, was admitted February 1st, 1897, under the care of Dr. Goodhart (clinical clerk R. H. J. Swan) for cough and shortness of breath. His illness commenced about 20th December, 1896, with an "attack of shivering," and he commenced coughing. The coughing continued and a few weeks later his legs began to swell. His face then swelled slightly, but afterwards the swelling went down. During the second week of the coughing he spat up some reddish brown blood. He had dyspnœa on exertion, but no palpitation. There was a history of chorea four years ago, for which he was treated at out-patients, and of pains in the joints during the last two years. His mother was suffering from rheumatic fever at the time of admission, but otherwise the family history was good.

Condition on admission.—Temperature 98.4°, pulse 134, respiration 36. Patient was well nourished, his cheeks were flushed and he was slightly jaundiced. There were slight choreic movements of the upper lip. The legs and ankles were œdematous. *Circulatory system.*—Pulse full, irregular, easily compressible and markedly dicrotic. The cardiac impulse could not be seen but was felt in the fifth space 12 millimetres internal to the nipple line. There was a presystolic thrill. A rumbling presystolic bruit was heard at the apex, a soft, blowing systolic murmur traceable outwards to the axilla, and these were followed by a sharp second sound. Above, the cardiac dulness

reached to the lower border of the third rib, internally it was limited by the mid sternal line, while it extended outwards for 11 millimetres beyond the nipple line. *Respiratory system.*—Impaired resonance posteriorly at both bases, where there were some râles and occasionally rhonchi heard. *Alimentary system.*—Tongue furred, abdomen rigid, liver dulness extended from sixth rib to four centimetres below the costal margin in the nipple line. The front of the abdomen gave a tympanitic note on percussion. In the flanks the resonance was impaired. No thrill could be obtained. *Urine.*—Acid, sp. gr. 1012. Albumen absent. On February 4th, cardiac impulse seen, but no thrill felt. Râles, rhonchi and diminished resonance at the right base posteriorly. *A faint soft diastolic murmur was heard in the second left space and could be traced downwards and outwards for about three centimeters* (Dr. Goodhart). On February 6th, the œdema of the legs had disappeared. The abdominal wall was still rather resistant. On February 9th, urine acid, sp. gr. 1020, a trace of albumen present. On February 12th, rhonchi heard all over chest. On February 15th, a reduplicated second sound was heard at the base. On February 16th, urine, sp. gr. 1024, acid; small amount of albumen present. On February 17th, heart-sounds very irregular. Presystolic and systolic bruits at the apex and a reduplicated second sound at the base, heard best at the junction of the third left cartilage with the sternum. Occasionally the sounds presented a triple character. Venesection was proposed but not carried out. On February 18th, a systolic bruit was heard at the right of the lower end of the sternum. The heart sounds were extremely irregular, the beats often occurring in groups of three, and occasionally a beat was missed. On February 20th, the temperature was 100·6°, pulse 120, respiration 60. On February 22nd, an increase in the area of cardiac dulness was noted. Above it extended to the lower border of the second costal cartilage, externally to 13 millimetres beyond the nipple and internally it was limited by the right margin of the sternum. Systolic and presystolic bruits were heard at the apex, but at the base the sounds were normal. Dulness and deficient entry of air at the left base posteriorly, was noted. On February 23rd, great dyspnœa and much coughing. Rising temperature. Temperature (6 a.m.) 100·6°. On February 26th, very severe dyspnœa. Systolic bruits at the apex and over tricuspid area. Slight œdema of the feet and legs (2 p.m.). Respiration 76, pulse 138. Paracentesis thoracis 227 cubic centimetres of greenish fluid withdrawn from the left chest. Sp. gr. 1012, containing twenty-eight parts per thousand of albumen, 0·1 per cent. of urea. The fluid coagulated on cooling. On March 27th, he was still dyspnœic (2 p.m., respiration 68, pulse 100). The air entry was better. Presystolic bruit at apex and systolic bruit at the lower part of the right side of the sternum. On March 1st, he was much better. Cardiac dulness less extensive; no tricuspid bruit. The sounds at the base were normal. On March 4th, presystolic and systolic bruits at the apex, soft systolic over the tricuspid area. Loud second sounds in the pulmonary area. On March 5th, soft systolic murmurs heard over the aortic and pulmonary areas. On March 10th, systolic and mid-diastolic bruits at the apex. Systolic murmur in the tricuspid area. Second sound accentuated over the pulmonary area and the first sound of a loud "scratching" character. General condition improving. On March 17th, cardiac impulse in the fifth space 13 millimetres internal to the nipple Dulness extends 2 centimetres to the right of the sternum. Systolic and mid-diastolic murmurs at the apex;

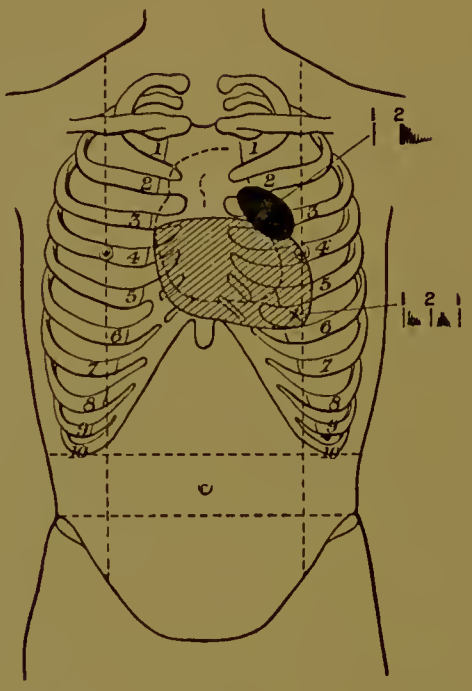

Fig. 11.

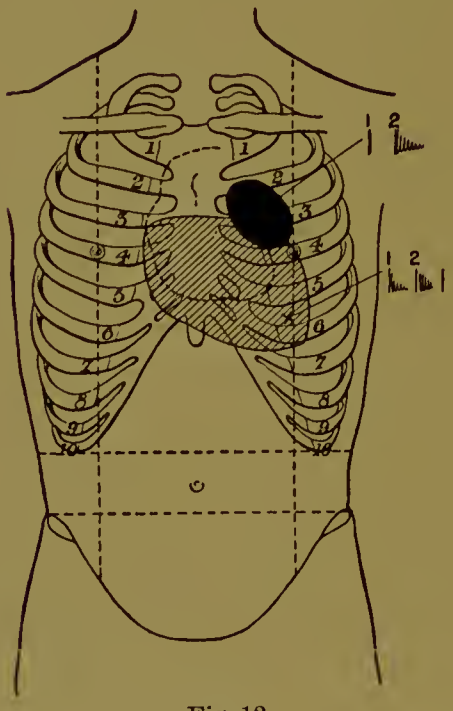

Fig. 12.

none over the tricuspid area. Both sounds loud and the second thickened over the pulmonary area. *At the junction of the second left costal cartilage with the sternum and traceable downwards and outwards half-way towards the left nipple a short, soft diastolic murmur was heard.* On March 18th, the pulmonary diastolic bruit was much better heard over the same area. A triple sound occasionally heard as if the first sound was reduplicated, but no reduplication can be heard at the apex. Patient better and getting up after dinner. On March 20th the pulmonary diastolic bruit was more distinct. On March 22nd the pulmonary diastolic bruit was traceable slightly upwards and outwards towards the left shoulder as well as towards the left nipple. Systolic bruit again heard in the tricuspid area. Bruits at the apex as before. The pulmonary diastolic murmur was not heard on the 24th, but was noted again having the same character and distributiou on March 26th and 29th (*vide* diagram). After this it was not again heard. Patient went out relieved on April 8th.

Case 16.—E. B., æt. 19, general servant, was admitted 15th March, 1895, under the care of Dr. Shaw (clinical clerk R. Mathias) for heart diseasc. She had been in St. Thomas's Hospital nine years before (1886) for chorea. She suffered from breathlessness and cough in the winter of 1893–4, and had had influenza and "inflammation of the lungs" about Christmas, 1894. She had only followed her present occupation for three months; prior to that she had always lived at home. She had had influenza again three mouths before admission, and since then she had had a cough and had suffered from breathlessness, precordial pain and palpitatiou after meals, but her appetite had becn good. Her legs and face had been swollen about March 1st—a fortnight before admission. She had not menstruated for three months prior to that she had been regular. Her mother had suffered from rheumatism, but otherwise the family history was good.

Condition on admission.—Temperature 98·8°, pulse 74, respiration 20. She was short and well-nourished; there was no œdema. *Circulatory system.*—The impulse was diffuse and felt in the fifth space as far out as 3·5 centimetres beyond the left nipple line. There was a thrill. Thc cardiac dulness commenced above at the third rib and extended a few millimetres to the right of the sternum. Presystolic and systolic bruits were heard at the apex, and systolic bruits were heard in the pulmonary, aortic and tricuspid areas. In the pulmonary area the second sound was accentuated. The pulse was small, regular and compressible. The respiratory system was normal; there was no cough. She complained of a pain in her side but no abuormal physical signs were detected. The urine was acid, sp. gr. 1014, and contained no albumen. The abdomen appeared normal. On March 17th, in addition to the other bruits, *a diastolic murmur was noticed in the second and third left intercostal spaces* (Dr. Shaw) (*vide* diagram). *The same diastolic murmur*

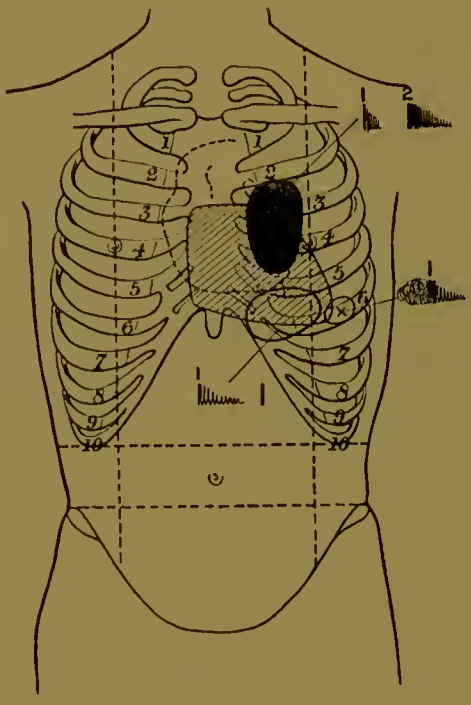

Fig. 13.

was also heard March 20th (J. H. B.) On March 23rd it was not audible (Dr. Shaw) and it was not noted again. Patient's condition improved and she went out on April 4th.